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Listerian Oration.[1]

NEPHRITIS.

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ALLOW me to thank the South Australian Branch of the British Medical Association for the signal honour of the invitation to deliver the Listerian Oration for 1932.

I have some trepidation at the sound of the word oration, since I am not about to deliver a formal ceremonial address. Nor have I anything to offer of original research. I have contented myself with choosing for a general critical survey a subject of wide interest to the general practitioner of medicine —nephritis—the various bilateral non-suppurative diseases of the kidney.

[1] Delivered at Adelaide, May 26, 1932.

The study of classification of kidney diseases commences with the work of Richard Bright, who published the first of his series of observations in 1827, the year of Lister's birth. It is with an apology that one ventures upon the oft-trodden ground of the classification of Bright's disease, but, as Osler pointed out, it is a good thing occasionally to pause and take stock of our knowledge. Certainly the importance of the kidneys as the seat of disease has not been under-estimated since Bright's time, but when one considers the original description and speculations of Bright himself, it cannot be claimed that increase in knowledge has been very fast, although the number and volume of opinions and investigations is enormous.

Much misapprehension exists as to the work of Bright. He was in reality the first to separate out of a large number of conditions with anasarca a group in which albuminuria was present. From that date there have been recognized different types of the disease and a host of writers have classified them

from one standpoint or another, from time to time, so that the state of mind of the student who sought a view with clear outlines was usually one of the utmost confusion. Confusion still exists, but the work of the last few years is clearing it away, and the practitioner can now approach the problem in his patient with a more intelligent view of the natural history of the disease and a more confident feeling as to its nature, its origin and its end. Indeed, our knowledge remained much the same for nearly a century since Bright, until Volhard and Fahr, in 1916, published their classification.

There are many who are bored with classifications. They regard them as of academic interest only. But this is wrong, surely. For my own part, I am not of those who love classification for its own sake, since I believe that one may lose sight of the real nature of disease in attempting to subdivide too much. But when one name covers a variety of diseases different in their origin and course, if not in their termination, a proper classification is imperative. It has a wider use than in the lecture room, for if it expresses the facts in bold and consistent outlines, it carries in itself a recognition of the disease we find in the individual patient, without which no effective treatment and no sound attempt at prognosis can be made. The ideal classification, then, is one which expresses, as simply as possible, fundamental distinctions only. The names that are used in such a classification are important, of course, but, as Volhard remarks, it is more essential to attain clarity concerning the nature of the disease than to be meticulous concerning the etymology of its name.

I think it probable (since it has been my own experience) that many of you have passed through various phases in your views on nephritis. At first, under the dominance of the pathologist, one imagined that one could visualize the pathological anatomy of the kidney of every patient in any stage in the course of the disease. But the *post mortem* room on many humiliating occasions proved the error of this belief. In Rowntree's words:

> There are many terms constantly used to indicate the various types of kidney found in nephritics, and one hears of small white kidneys, large white kidneys, small red kidneys, large red kidneys, and yellow kidneys, until one may be forgiven if he becomes confused; we do well to remember the extreme uncertainty of predicting the kind of kidney from the clinical symptoms and signs, and the habit of referring certain signs to a "large white kidney" or some other such kind is to be discouraged.

One turned then to a method which was simplicity itself. Leaving the pathological anatomist to his own devices, many were content with a division into acute, subacute and chronic nephritis, sometimes with œdema, sometimes with hæmaturia. How unsatisfactory was this when one came to treat a patient and to advise him on his future! One could not but be harsh in treatment or uncertain and gloomy in outlook.

The physiologist then took a hand and one was pleased to feel that much of the real nature of these diseases was now clear. Here one recognized the patient with hydræmic, there the patient with azotæmic, nephritis. There existed but three types: salt nephritis, water nephritis and nitrogen nephritis. It was delightfully simple, in spite of the fact that the types seemed frequently to get mixed. Soon, however, without disparaging the value of the facts on which this subdivision was based, the conclusion was reached that the matter was not so simple and that the differentiation on these lines was not a true one. The biochemist gives us valuable information in treatment and prognosis, but he is not the final arbiter in classification.

The older pathologists laboured under the difficulty that their observations were made at one moment of time in each patient, and that the moment of death. But different diseases progressing by different pathways may lead to the same terminal condition, and it is the various steps in the pathway, rather than the ultimate end, which the clinician wants to know. Latterly, some classifications have come from brilliant pathologists, principally in London. They lack nothing in meticulous detail; indeed, it is a feat to remember them. But they are of little help to the practitioner.

The pathological-physiological classification promised much in its simplicity. It appears, however, that no physiological classification of nephritis is possible until we know more about the normal physiology of the kidney.

> The daily performance of the healthy kidney is no doubt a combination of mechanical and vital processes. Yet the more one follows the conflict of evidence and the varying results that have been obtained by the use of similar experiments on kidney functions, the more must it be realized that these functions need not be absolutely hard and fast, unalterable, specific rules of procedure. Glomerulus and tubule assist one another; in time of stress, one may compensate for failure of the other; if both fail together, their work may be temporarily performed, in some degree, by the adjuvant systems, the alimentary canal, the skin and the other lesser excretory organs. The variations in experimental results and the difficulty experienced in trying to lay down hard and fast rules as to kidney function arise from this very "give and take" between the different parts of the kidney and between the kidney and its fellow organs.

Further:

> As yet, almost nothing is known of the pathological physiology of the kidneys. It is fascinating, however, to speculate on what happens when the rate of flow is changed by vaso-constriction, hypertensive disease or cardiac weakness; what changes take place when the small renal vessels become sclerosed and the blood supply to the glomerular membrane and the renal epithelium decreases; what happens when toxins affect these structures.

What is the nature of the substance which, circulating in the blood, causes those symptoms of so-called "uræmia", which are not due to associated vascular changes? These are problems as yet unsolved, and consequently no satisfactory physiological classification is yet possible.

Of late years, however, a classification based mainly on clinical data has emerged as the result of the labours of Volhard and Fahr in Germany, supported by the work of Addis, of Chicago, and Van Slyke and his associates in the Rockefeller Institute in America. In this classification the outlines are clear and consistent, and it is the most helpful and

practical method for the practitioner in the ordinary routine of practice. This is based on the study of the clinical course of the diseases primarily, but this has been correlated to the pathological findings. For the first time the clinically defined conditions have been related satisfactorily to anatomical changes in the kidneys. The classification is imperfect necessarily in the present state of our knowledge, but in that it is related to the clinical course, it is more satisfactory than any other.

I propose to examine this in the light of my own experience and to point out in what respect I think it helps us as general practitioners.

Approaching the problem from very different directions, these authors have arrived at conclusions which are in the main identical. The disease nephritis embraces three different kinds of disease process, which are characterized clinically by:

1. Hæmaturia, acute, intermittent or chronic, with varying degrees of œdema, hypertension and nitrogen retention. The fundamental pathological change in this type is inflammation of glomeruli. The disease is named hæmorrhagic or glomerular nephritis.

2. Hypertension, which is followed by serious kidney disease. The pathological change is thickening of the renal arterioles. The disease is named nephrosclerosis or arteriosclerotic Bright's disease.

3. Œdema and marked albuminuria with no hæmaturia, hypertension or nitrogen retention—primarily due to degeneration mainly of the tubules. This disease is spoken of as degenerative Bright's disease or nephrosis.

Glomerular or Hæmorrhagic Nephritis.

In the acute stage glomerular or hæmorrhagic nephritis is a very distinct and familiar condition, a clear-cut clinical entity, sudden in onset, and associated usually with obvious infection; hæmaturia and albuminuria are its outstanding features, usually associated with œdema of varying degree. There may be some disturbance of renal function, some rise of arterial tension and some anæmia. In severity it ranges from the mildest of attacks from which recovery is complete and sometimes rapid, to a severe debilitating disease which leads inexorably to death. Considerable variation in the clinical manifestations of the disease is observed, but always there is hæmaturia which is frequently gross and macroscopical, although sometimes the hæmorrhage is discoverable only on examination with the microscope. The urinary sediment shows invariably the presence of red blood cells and sometimes of blood casts.

It is now almost universally recognized that the cause is a toxæmia, due to infection with the streptococcus.

If the attack proceeds to cure, the clinical manifestations disappear in turn. No set rule may be recognized, but usually hæmaturia of gross type is the first to go, and the last to disappear is albuminuria, but in other instances microscopical hæmorrhage or a tendency to œdema may be the last sign to be noticed. Sometimes, although rarely, all symptoms and signs of the disease may disappear in a few days. Indeed, I am convinced that not infrequently it escapes notice when the symptoms are slight and the more obvious signs transient. Usually, however, the duration of the disease, when its clinical manifestations have been very definite, is a matter of several weeks. Van Slyke and his associates found in the series of patients investigated by them that usually noticeable improvement began within four months in all patients who were destined to recover; and they note further that in some the disease lasted for as long as fifteen months before all symptoms and signs of the disease had disappeared.

Complete recovery may take place, even if marked decrease in renal efficiency, anæmia and hypertension are present. It is important to recognize this, because in contrast with the later "active chronic stage" of the disease, these signs may not have such grave prognostic significance. I believe that if the source of infection is completely removed, a streptococcal infection of the tonsils or teeth for example, recovery frequently takes place from the acute stage, even though at the height of the attack renal efficiency tests reveal unsatisfactory function, or marked rise in arterial pressure is present. On the other hand, an attack of slight apparent gravity or even an unrecognized one may not proceed to cure, but may lead on to the "active chronic stage", in which the prognosis is bad. Probably, when this happens, a continuing infection is present. It is to be clearly recognized that the most important point in treatment is the removal of all infected foci from which toxæmia may arise. I realize that in these days focal infection is a veritable journeyman of medicine, since there is a tendency in many to attribute every disease whose cause is not immediately obvious, to infection of teeth or tonsils or other less accessible organs. How many patients, victims to this fashion, find themselves denuded of useful and innocent parts of their anatomy without amelioration of their disease! But in this case we are on sure ground and much suffering and many lives may be saved by the recognition of the streptococcal origin of glomerular nephritis.

In many instances, however, when the acute attack is of great severity, it sometimes proceeds to a fatal termination within a few days or weeks only.

In a large number of patients, however, the disease does not proceed to cure or death immediately. It gradually develops into a chronic stage in which many diverse associations of signs may be observed.

As indications of prognostic value in acute glomerular nephritis, it is to be recognized that patients showing a high initial degree of albuminuria, hæmaturia, œdema or defective nitrogen excretion, may recover or pass into a latent stage in which a life of normal activity is possible.

The disappearance of hæmaturia as the disease progresses is a favourable sign, but its persistence

and amount do not appear to bear a definite relation to the severity or rate of progress of the disease.

The degree of albuminuria at onset has no prognostic significance, but its diminution or disappearance is a favourable sign (if other indications are favourable). Apart from this, the continuance and degree of albuminuria have more significance in diagnosis than in treatment or prognosis.

A marked diminution in nitrogen excretion at the height of the initial attack does not indicate necessarily a grave prognosis, but a persistence of defective function with no indication of improvement after four months indicates (Van Slyke) the development of the disease to the active chronic stage, from which ultimate recovery is impossible.

Similarly, patients who recover completely may have shown marked hypertension or œdema during the acute attack, but the persistence of one or other is a grave sign, and, with defective renal function, hypertension and œdema are more important prognostic criteria than hæmaturia and albuminuria.

Addis has expressed the course of the disease in the manner shown in Figure I.

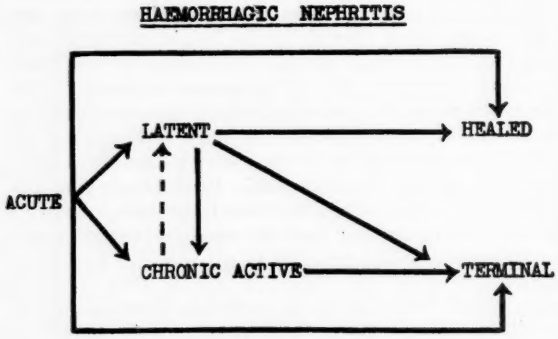

FIGURE I.

The most valuable feature of this scheme of classification is the recognition of the latent and chronic active stages, because, under these names exist a large number of conditions apparently so different as to have been regarded as separate entities. It is important to recognize that they are not fundamentally different and to study how they arise. Hæmorrhagic nephritis is a disease in which the essential pathological lesion is a destructive inflammation of the glomeruli. Its essential clinical sign is hæmorrhage, and albuminuria is always present. If complete recovery or death does not ensue, the disease may become "latent", that is, the patients may partially recover. In this state there are no symptoms of disease, but examination, revealing some albuminuria or some slight inconstant hæmaturia or some diminution of functional renal efficiency, shows that the disease has not disappeared. It may ultimately disappear, and indeed frequently does. In such case, the slight signs of abnormality disappear one by one. A persistent renal deficiency of some months' duration, a definite persistent hypertension or a definite anæmia is never found in the latent state. The absence of these signs warrants a hopeful prognosis, if all infection is removed and fresh infection does not occur. Frequently, however, the latent stage, through continuing infection or through fresh infection, passes into the chronic active stage. But more frequently the chronic active stage develops directly from the acute.

The chronic active stage merits study in some detail. The phenomena of glomerular nephritis are: hæmorrhage, albuminuria, diminished nitrogen excretion, œdema (dependent on deficient plasma protein), hypertension and anæmia (see Figure II). In the chronic active stage hæmorrhage is always present, although often it is intermittent. Thus the microscope will reveal blood cells in the urinary sediment if several specimens are examined, especially if Addis's technique of reducing the fluid intake to the minimum is followed. Proteinuria is also invariable and some diminution in the capacity of the kidney to excrete nitrogen. The albuminuria, although usually marked, may in some instances be slight, but as a rule it is more pronounced than in nephrosclerosis. The deficiency in nitrogen excretion also varies within wide limits, but it invariably increases as the disease runs its course. At times it has been my experience that the blood urea and urea concentration test (Maclean) did not reveal a clear undoubted defect at first, although this appeared later.

Van Slyke has stated that his method of measuring the blood urea clearance is the most valuable means of determining renal functional efficiency. In this the number of cubic centimetres of blood per minute cleared of urea by renal excretion is expressed. In Sydney we have had some experience if this method of investigation in the hands of Hansman, who concluded that Maclean's method (which is in general use in our State) of measuring the concentrating capacity of the kidney is of greater value and reliability in clinical application in early stages of defective function.

No criterion of progress is superior to this one. A continuing diminution of the power to excrete nitrogen is indicative of the increase in glomerular destruction and has but one melancholy significance.

To this triad of hæmaturia, albuminuria and deficient nitrogen excretion are added other elements in varying degree, sometimes slight, sometimes the reverse.

In one patient hypertension is outstanding, in another œdema, and in a third anæmia. And in some patients all these signs coexist—a frequent occurrence. Thus it comes about that a very varying clinical picture may be presented by different patients in the chronic active stage of glomerular nephritis. These conditions are not different clinical entities, they all spring from the same stem (Figure II).

As illustrations of varying syndromes, some actual records are of interest.

J.H., aged fifty-one years, a bank manager, complained that for nine months he had noticed progressive weakness, a vague feeling of malaise, with capricious nausea. He had been examined on various occasions, but no definite

diagnosis had been made. On admission to hospital, he was of healthy appearance. Very advanced apical sepsis was present in several teeth, as revealed by radiographic examination. The cardio-vascular system showed no abnormality. The peripheral vessels were soft, there was no hypertension and the vessels of the eye grounds were normal. There was no cardiac hypertrophy and no œdema. The blood count was normal. The urine showed a faint trace of albumin and red blood cells in excess of the normal, and a few casts (granular and hyaline). The fasting blood urea was 398 milligrammes *per centum*. There was no previous history of illness of any kind nor of an acute onset.

This patient's condition slowly deteriorated. The asthenia and nausea increased, while the blood urea slowly rose. A microcytic anæmia developed. Ultimately, stupor appeared, followed by coma, and the patient died in "uræmia".

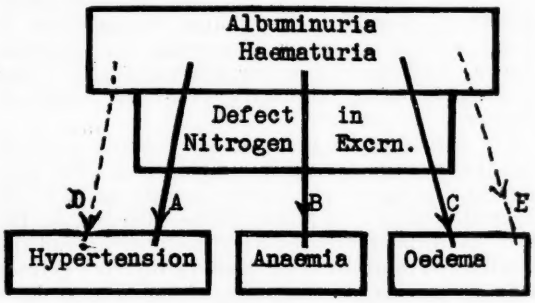

FIGURE II.

In this figure a graphic representation is made of some of the syndromes found in active chronic glomerular nephritis. Commonly all five factors are present, but at times are found (*a*) albuminuria, hæmaturia, defective nitrogen excretion and hypertension, (*b*) albuminuria, hæmaturia, defective nitrogen excretion and anæmia, (*c*) albuminuria, hæmaturia, defective nitrogen excretion and œdema, but rarely (*d*) albuminuria, hæmaturia and hypertension, and (*e*) albuminuria, hæmaturia and œdema alone occur in patients with chronic glomerular nephritis. If these syndromes do occur, they are of very short duration, signs of defective nitrogen excretion soon making their appearance (*A* and *C*).

This is an instance of a somewhat uncommon type, in which the triad of albuminuria, hæmaturia and defective renal function existed alone, and death occurred as the result of a pure renal insufficiency. More commonly, anæmia appears as such a type progresses, but it is not invariable (B, Figure II).

The consideration of the symptoms presented by such a patient indicates the effect of insufficient function undisturbed by associated vascular changes such as hypertension, cardiac hypertrophy *et cetera*. These symptoms may be compared with the phenomena seen in complete anuria of "reflex" or obstructive origin, in rare instances where a single kidney has been removed (the other kidney being congenitally absent), and in some poisonings. From this it appears that renal insufficiency gives rise to no striking symptoms until a terminal stage of stupor occurs, followed by coma and rapid death. Beyond general asthenia, slight nausea and discomfort, no symptoms suggest alarm until stupor appears. There are no vertigo, blindness, severe and intractable vomiting, convulsions, and convulsive twitchings, Cheyne Stokes breathing, secondary inflammations of mucous membranes and cerebral œdema, such as mark so many instances of so-called "uræmia". Indeed, as Mosenthal has suggested, the time is ripe for the term "uræmia" to be discarded, since it has no definite connotation, the units of this complex arising mainly from associated vascular changes and not from a failure of renal function which the term suggests.

E.S., aged thirty-seven years, a merchant, suffered from a severe attack of tonsillitis one year before presenting himself for examination for life insurance. Then a definite hypertension was discovered, with albuminuria. Further investigation revealed the presence of an abnormal number of red cells (the technique of Addis being used) and a slightly raised blood urea content, with slightly diminished power of concentration. The tonsils were found to be grossly infected with streptococci. No anæmia or œdema was present.

Here the triad was accompanied by hypertension, which on superficial examination dominated the picture (A, Figure II). The details of the examination left no doubt that this was not an instance of nephrosclerosis.

A common experience is to encounter cases of this type in which an erroneous diagnosis of arteriosclerotic Bright's disease has been made. In this latter condition there is never a history of hæmorrhagic onset, there is no hæmaturia, except in the terminal stage occasionally, and the hypertension definitely precedes the evidence of renal damage. Generally also, there is less protein in the urine. In this type death may come from a vascular catastrophe or from renal insufficiency.

Commonest of all syndromes in active chronic glomerular nephritis, however, is that in which to the fundamental triad of symptoms is added œdema (C, Figure II).

"Œdema is usually an outstanding characteristic in the chronic active stage, although a minority of patients do not show it." Sometimes it is always present, but frequently the patient hovers on the verge of œdema; on some days it is present, on others absent; and this often without obvious explanation.

When a patient has marked œdema and albuminuria, with but little nitrogen retention, a familiar picture is seen—the "nephrotic type" of glomerular nephritis. It is difficult to distinguish this from a pure nephrosis, if hypertension is not present and renal function not materially defective. The presence of red cells in the urine is the most "consistent single differential sign".

A history of an acute onset with hæmaturia is conclusive and, indeed, is at times the only means of differentiation. But differentiation is important because of prognosis. The nephrotic type of glomerular nephritis progresses inevitably (slowly or rapidly) to death. The patient with pure nephrosis may recover or may succumb to an intercurrent infection. It is probable that in the nephrotic type of glomerular nephritis a degenerative condition of the tubule has been added secondarily to the primary hæmorrhagic lesion.

Anæmia is an indication of the toxic damage to the organism and is probably due to injury to the bone marrow. Of grave prognostic significance, it usually occurs late in the chronic active stage. It does not occur when the excretory capacity of the

kidney is undamaged, but the reverse is not true. So it comes about that a patient may progress to his death from "uræmia" with a normal blood picture. The usual sequence is that a deficient renal capacity is well in evidence before a definite anæmia appears (B, Figure II). When a definite anæmia is present, the prognosis is grave.

Terminal Stage.—The terminal stage is not clearly demarcated from the previous stage. Van Slyke adopts an arbitrary figure of blood urea clearance as indicator of the terminal stage. In general it is characterized by increasingly defective renal function, as the glomeruli become completely disabled. Hæmaturia is slight as the number of live glomeruli diminish, anæmia increases, œdema, if present, is mainly cardiac, the blood pressure often rises still further and anæmia sometimes becomes extreme. If a vascular catastrophe does not cause death it comes in "uræmia".

Degenerative Nephropathy or Nephrosis.

There is more confusion about degenerative nephropathy than in the other two of Volhard and Fahr's classification.

In it are included the degenerative kidney conditions due to a heterogeneous set of intoxications and infections, such as those of pregnancy, syphilis, tuberculous and long standing staphylococcal infections (amyloid degeneration), and the disease called variously "genuine", "true" or "pure" nephrosis or "cryptogenic" nephrosis.

As Leiter observes, "nephrosis began its existence in medical nomenclature as a point of view". It arose from the need for a general term to indicate those common kidney changes which are not due to a true inflammation or nephritis or to primary vascular changes. Muller suggested the term "nephrosis" in 1905, to be applied to those conditions of the kidney which were degenerative in origin (where the degeneration was not secondary to vascular disease). Shortly the term became popular, although combated by the pathologist. But it took on a different significance in different hands and to some extent in different countries. Latterly, however, the tendency has been to restrict the use of the term more and more, so that in the practice of the majority it now indicates the true or "genuine" nephrosis. I believe that this is wise. There are still some who deny the existence of this as a distinct clinical and pathological entity, but I am convinced they are not right. Certainly a diagnosis of nephrosis is frequently made on clinical grounds alone, and the passage of time reveals the error, the patient having been affected with the nephrotic type of glomerular nephritis. But in other instances the clinical features, the biochemical findings and the autopsy findings are so consistent as to leave no doubt that a pure nephrosis is a definite entity. The name itself is unsatisfactory, but no better name has yet been suggested. When properly used and understood, the term is a valuable one and, in the present state of knowledge, necessary.

Nephrosis is a rare chronic disease of children and young adults, insidious in onset and of unknown causation. That certain infections, such as syphilis and upper respiratory infections, predispose to it, is possible, but they are not its prime causes. As positive findings there are heavy albuminuria, œdema, usually massive in degree, marked pallor and a typical urinary sediment. The "negative" findings are absence of hæmaturia, of vascular changes, and of diminished renal efficiency. It runs a chronic course, interrupted by exacerbations due to no obvious cause. Complete recovery may ultimately occur, but usually death results from some intercurrent infection, to which there is a remarkable susceptibility. At *post mortem* examination a characteristic tubular degeneration is found without primary glomerular or vascular change. Occasionally minor grades of glomerular involvement of a secondary type are found at autopsy.

The actual cause is unknown. Active syphilis appears sometimes to play a minor rôle. The *post mortem* findings reveal kidney changes only, which suggests that it is a purely kidney disease; on the other hand, the decreased basal metabolism and the striking tolerance to large doses of thyreoid extract and the extrarenal character of the œdema have given rise to the theory that nephrosis is a metabolic disorder, the renal changes being secondary. Whatever its cause and whatever its nature, there is no doubt that a disease is encountered, very rarely it is true, characteristic in its clinical and pathological aspects and differentiated from other nephropathies after careful examination.

There is no doubt that many patients have been labelled as sufferers from nephrosis, when the condition was really active chronic glomerulonephritis. Pure nephrosis, confirmed at autopsy, is extremely rare, but the clinical syndrome, in all of its details, is vastly more common. How are we to differentiate it? In the first place, a previous history of nephritis with hæmaturia is almost conclusive that, whatever other conditions are present or absent in a patient with marked albuminuria and œdema, the disease is primarily glomerular in origin.

Although some authors state that neither normal renal efficiency nor complete recovery of the patient will exclude glomerular nephritis, I think it is a sound rule that when continual examinations do not reveal any deficiency of the power to excrete nitrogen, when hypertension and other vascular changes remain absent, and when hæmaturia is not revealed by the microscope, even under intensive conditions of examination, the young patient with heavy albuminuria and gross œdema during a long course is suffering from a pure nephrosis.

An exception to this occurs when an acute glomerular nephritis is proceeding to recovery and hæmaturia and defective function have disappeared, leaving only albuminuria and œdema. Again, a difficulty arises at times when, in the early stages of glomerular disease, the decrease in renal efficiency is very slight, so slight indeed that tests reveal no

definite evidence of urea retention in the blood or diminished capacity for concentration of urea by the kidneys. In such instances it is probable that time for observation of the course of the disease, as shown by tests of renal efficiency, is necessary before a definite decision can be made.

That an accurate diagnosis is of great importance is obvious when one recalls that nephrosis is a disease from which complete recovery sometimes occurs. I do not believe that the same can be said of the nephrotic type of nephritis. In the latter, after ups and downs it may be, death inevitably ensues, but in the genuine nephrosis, although there may be temporary apparent recovery followed by renewed activity, in some instances permanent recovery takes place. In others, infections by such organisms as the pneumococcus or staphylococcus are exceedingly common, and the majority of patients with this disease succumb to one or other of them.

A clinical picture similar to that of a true nephrosis is not infrequently encountered in the presence of some obvious infection. Active syphilis stands first among these, but, in addition, amyloid disease from whatever cause arising. It is probable that the nephrotic component is quite secondary, and the presence of evidences of active syphilis, chronic tuberculosis, osteomyelitis of long standing, or some other similar condition places the diagnosis beyond doubt. In these diseases recovery is impossible.

Nephropathies of Pregnancy.

There still remain to be mentioned the nephropathies of pregnancy. Much confusion exists as to the nature of these. Pregnancy may supervene on either the glomerular or the arteriosclerotic form and may activate or accelerate their progress. It is not uncommon to meet patients presenting the characteristic picture of an active chronic glomerular nephritis, the starting point of which appears at first sight to have been a pregnancy. The attribution of the disease to a "pregnancy nephritis" in such a case is erroneous. Similarly, arteriosclerotic Bright's disease, although it may first come under notice during pregnancy, never arises primarily as the result of pregnancy. There are, however, two conditions fundamentally associated with the gravid state, the "nephrosis of pregnancy" and the eclamptic toxæmias. Although they sometimes coexist, they are different in clinical characters and in pathogenesis.

The nephrosis of pregnancy occurs in the latter half of the term of gestation and differs in no essential factors from the other nephroses not termed "genuine", except in the fact that recovery frequently occurs.

Eclamptic toxæmia, on the contrary, is a state in which vascular phenomena, such as cerebral œdema and small hæmorrhages, absent in the nephroses, play a dominating part in the causation of the clinical picture.

Arteriosclerotic Bright's Disease or Nephrosclerosis.

Arteriosclerotic Bright's disease or nephrosclerosis is probably the commonest form of nephropathy. In the past, however, many instances of chronic glomerular nephritis have been placed in this category in error, so that its apparent frequency possibly has been overstated.

There are to be observed as its salient features a marked increase in arterial tension, with the resultant cardiac hypertrophy and a slight grade of albuminuria. Characteristically, hæmaturia is absent, also non-cardiac œdema. At first, if any defective renal function is present, it is slight. Anæmia is occasionally present, but to a much less degree than in hæmorrhagic nephritis.

The primary pathological changes are in the arterioles.

The disease may run a benign course over many years and death occur by vascular causes, the renal function being affected to a relatively slight degree. The great majority of patients with benign nephrosclerosis die of a vascular catastrophe in the vessels of the brain or heart.

In some instances, however, the benign disease rapidly changes to a malignant form, in which a severe and usually rapid deterioration in renal function occurs and dominates the clinical picture, and the patient dies of renal and not vascular failure, "uræmia". It is probable that in such patients a glomerular inflammation becomes superimposed on the fundamental arteriosclerosis. Frequently the sign of this change is the development of retinitis.

The recognition of this type of nephropathy depends on the course of the disease, the hypertension being the first and only sign usually for a considerable period, followed gradually by slight evidences of defective renal function. Albuminuria is usually slight in degree. Although a slight albuminuria only is seen sometimes in chronic glomerular nephritis, it is not the rule. Hæmaturia is never found under intensive conditions of examination, except in the terminal stage. This is probably the most valuable criterion in case of doubt. Bearing in mind the characters of chronic glomerular nephritis in which hypertension is marked and non-cardiac œdema is absent, namely, the degree of albuminuria, the presence of red blood cells in the urinary sediment and the evidence of defective renal function, there is little difficulty in differentiating this from a benign nephrosclerosis. Yet it has been my experience that considerable confusion of these two nephropathies exists. This is of particular importance, since the prognosis in regard to time may be very different, nephrosclerosis of benign type running a much slower course than chronic glomerular nephritis.

When the malignant phase of the disease is present, there is no difficulty in differentiation if the course of the disease in the patient has been observed. But greater difficulty may be encountered if the patient first comes under observation when the malignant phase is well established and renal failure is present. Here again, however, the pre-

sence of hæmaturia is a valuable criterion, since this condition is present only as a terminal one in nephrosclerosis.

Conclusion.

In this review I have attempted to give a description of the nephropathies, in which the various types have been given clear and consistent outlines. There will come to the minds of most of you, no doubt, many patients who do not fit in easily to a definite subdivision. This is likely in any scheme, but, I think, less in this than in any other. Certainly the stages of glomerular nephritis are not sharply defined; there is no single sign whose appearance determines that at some definite date, some finite period, the acute stage becomes the latent, or the active chronic, or that the benign type of nephrosclerosis has become malignant. Nevertheless, these stages are clearly recognizable when established. Also the sharp edges of the picture are often somewhat blurred in the "mixed types", so-called, but these matters are secondary in importance to definite conceptions of the pathogenesis, the course, the time relations and the prognosis of one of the largest, most frequent and most important groups of diseases which we are called upon to treat.

RIGHT-SIDED ABDOMINAL PAIN.[1]

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THE title of this paper covers a wide ground, so wide that to attempt to deal with it adequately in the time available would be impossible; so wide that I intend to confine myself mainly to various possibilities rather than to go deeply into treatment, which, in most cases, is well understood.

The differential diagnosis is the most important factor of this oftentimes very difficult and troublesome symptom.

It is well to consider separately the acute and chronic manifestations of right-sided abdominal pain, but naturally there are many borderline or subacute cases.

ACUTE CONDITIONS.

If in the acute class we are spared many of the difficulties of diagnosis of the chronic, we meet with other obstacles, such as (*a*) the extremely serious condition of the patient, which may prevent an accurate history being obtained; (*b*) the diffuseness of the tenderness, and perhaps the presence of rigidity, which may prevent an accurate localization of the primary lesion; (*c*) the urgent need for operation which prevents the thorough examination possible in the chronic condition. Not infrequently a mid-line incision has to be made for exploratory purposes.

Before discussing those possibilities of acute right-sided pain resulting from disease, it would be well to dispose of those due to trauma.

[1] Post-graduate lecture delivered at Sydney on May 30, 1932.

Traumatic Causes.

The result of trauma is either: (i) hæmorrhage from liver or kidney or vessels of the mesentery, or from a ruptured Graafian follicle; or (ii) perforation of a hollow viscus.

Hæmorrhage into Peritoneal Cavity.

Hæmorrhage into the peritoneal cavity will give rise to signs of movable dulness in the loins, but even though there be considerable effusion of blood, yet this sign may be uncertain. The blood will early make its way to the pelvis and float the intestines up on it. Pain is not infrequently referred to the shoulders. There will be in many cases quickening of the pulse, but the rate sometimes remains normal; the temperature will be subnormal at first, and later will show moderate elevation.

This condition is often slow in onset, which sometimes puts one off one's guard; the degree of trauma may also be thought to be insufficient to produce it. Blood acts as a peritoneal irritant and causes muscular rigidity.

Hæmorrhage into the peritoneal cavity does not by any means always call for operation; it not infrequently ceases owing to intraabdominal pressure, and is gradually absorbed. These cases require much judgement and watching.

If the hæmorrhage is from the liver, I think that in most cases it is better not to operate; the hæmorrhage will usually subside with absolute rest. If the abdomen is opened, sutures will not readily control the laceration; plugging with gauze will be necessary, and this is not the most desirable procedure, since it may be followed later by secondary hæmorrhage.

If the hæmorrhage were known to come from the spleen, I should strongly recommend splenectomy, because of the great likelihood of recurrence, even after absolute rest.

If the right kidney is affected, then there will be some rigidity and abdominal distension, suggesting an intraperitoneal lesion. Fortunately, hæmaturia puts us on the right track.

In passing, I may say that not every case of ruptured kidney can be treated conservatively; early nephrectomy, before the patient has lost too much blood, is sometimes desirable.

Rupture of Hollow Viscus.

If rupture of the hollow viscus occurs, it is generally the fixed portion of the duodenum that ruptures. On the left side it is usually the jejunum near its origin. The temperature is subnormal at first; in twenty-four hours it may be normal and then begin to rise. The pulse rate also is deceptive. For thirty hours it may not rise to any great degree. Often, by the time the temperature and pulse rate have sounded the alarm, the time for successful operation has passed. There may be some loss of liver dulness, but this also is a rather unreliable sign. There will generally be, from an early hour, some degree of muscular rigidity, most marked over the site of the rupture.

Later on, often when it is too late for successful operation, come the signs of peritonitis. One of the least often recognized of these signs is pain about the top of the right shoulder, due to inflammation of the under-surface of the right diaphragm. In the hope that the case is not serious or that it is one of hæmorrhage from the liver, the surgeon is inclined at times to leave it alone until, too late, he realizes the necessity for intervention. The lesson is, when in doubt, to do an exploratory laparotomy.

Thoracic Injuries.

The surgeon is frequently left in doubt as to whether the injury is thoracic or abdominal. A false step in treatment may endanger the patient's life. If the injury is thoracic, morphine and rest in the Fowler position will be indicated. A needless operation may be fatal from post-anæsthetic complications superimposed upon the thoracic injuries.

Some of the pain may be referred to the abdomen; the accompanying shock may lead even an experienced observer to implicate the abdomen. In twelve hours or so one should be able definitely to distinguish a thoracic from an abdominal injury, but it is not to be forgotten that the two may coexist.

Non-Traumatic Causes.

Some of the affections causing acute right-sided pain are: (i) cholecystitis, (ii) ruptured duodenal or gastric ulcer (at operation it is often difficult to say on which side of the pylorus the perforation is), (iii) appendicitis, (iv) acute pancreatitis, (v) acute salpingitis, (vi) ruptured ectopic gestation, (vii) torsion of Fallopian tube, (viii) ptomaine poisoning, (ix) renal affections, such as renal colic and pyelitis, (x) torsion of small intestine, (xi) chest conditions, (xii) Henoch's purpura, (xiii) thrombosis of branch of superior mesenteric artery.

Cholecystitis.—The pain and constitutional symptoms accompanying the biliary colic of cholecystitis are sometimes so considerable that for a time a doubt may arise as to whether or not the case is one of perforated ulcer. The failure of the signs and symptoms to become more serious as the hours go on excludes the possibility of a perforated ulcer. In gall-bladder disease there is a marked tendency for the pain to extend from the right subcostal margin to the right infrascapular region with accompanying hyperæsthesia of the skin; the rigidity is not usually so board-like. Pain and hyperæsthesia at the top of the shoulder are not present; they are a text book myth in most cases. Pain referred to the left side of the abdomen in the left hypochondriac and epigastric regions is occasionally present, being due to reflex distribution along the eighth dorsal nerve. There is usually a preceding history of a sense of fullness after meals.

There is seldom harm in waiting in a case of cholecystitis; in fact, it is safer to operate on the patient between the attacks; there are seldom fatal complications such as may ensue from an inflamed appendix that is left. At the same time, the diagnosis is made clearer by waiting.

Perforated Ulcer.—In the diagnosis of perforated ulcer a previous history of indigestion is helpful if the patient is well enough to give it to you, which he is not always; the indigestion is generally of the hunger pain variety. The extravasated contents early make their way down the right kidney gutter towards the caecum and appendicitis is not infrequently diagnosed. The rigidity, though universal on the right side, is usually more severe in the upper quadrant. Liver dulness is diminished in 50% of cases. Early shock is considerable, but it may be four or five hours or more before there is any noticeable increase in the pulse rate. The temperature is at first subnormal. With the onset of peritonitis there is top-of-the-shoulder pain.

Appendicitis.—In regard to appendicitis, there is not much new to be added, except to emphasize that marked muscular rigidity means gangrene or perforation, or a stage not far removed from them. This stage may be arrived at as early as twenty-four hours after the onset of the pain.

Acute Pancreatitis.—Acute pancreatitis is usually the cause of intense pain, which is felt not only in the upper part of the abdomen, but in one or both loins. It is often accompanied by delirium or strangeness in manner.

Acute Salpingitis.—In acute salpingitis the signs and symptoms are referred to the pelvis and lower part of the abdomen and are either Neisserian or puerperal in origin. The location of the pain is several inches below McBurney's point.

Ruptured Ectopic Gestation.—The pain of ruptured ectopic gestation is not infrequently referred to the liver region, being due to the blood travelling up there and irritating the under-surface of the diaphragm.

Torsion of the Fallopian Tube.—Torsion of the Fallopian tube is rare.

Ptomaine Poisoning.—I have known ptomaine poisoning to simulate acute appendicitis, but the history of diarrhœa and the affliction of others in the house will help to differentiate it.

Renal Affections, such as Acute Pyelitis and Renal Colic.—Pyelitis appears to be more common in women and children. Examination of the urine should reveal any implication of the urinary tract (naturally the specimen in women must be a catheter one). I have noticed on occasions a mild pyelitis accompanying a cholecystitis.

With acute renal colic right-sided rigidity is not uncommon, simulating for a time an intraperitoneal affection. Descent in the location of the pain (as the stone descends the ureter) is not uncommon in ureteric colic.

Murphy's first percussion sign is valuable in revealing renal affections when they are present in a marked degree, but it is not pathognomonic, for I have obtained it in cases of retrocaecal appendicitis.

Torsion of the Small Intestine.—Torsion of the small intestine is not common, but I have seen

several cases in which it simulated an acute appendicitis.

Chest Conditions.—The chief of the chest conditions are pleurisy and pneumonia. In these the pain is sometimes referred to the abdomen, along the distribution of the thoracic nerves, but seldom is the tenderness on palpation sufficient to justify the diagnosis of appendicitis or cholecystitis. The inability to detect pneumonic or pleuritic signs often leaves one in doubt, but there is often a dilatation of the *alæ nasi*, which puts one on the track.

Henoch's Purpura.—In Henoch's purpura there is a hæmorrhagic effusion into the wall of the bowel, with colic and perhaps (but this may come later) a purpuric eruption. I have seen one case.

Hæmatoma in the Right Rectus Muscle.—Hæmatoma in the right rectus muscle may be a result perhaps of insignificant trauma. It simulates appendicitis.

Mesenteric Suppuration.—Suppuration in the mesentery or in the retroperitoneal tissue (as a result of infection from the bowel) I have known to simulate appendicitis until laparotomy revealed the cause.

Intussusception.—Intussusception, of course, will early be thought of in children.

Chronic Conditions.

If in chronic cases we have more time for reflection, we certainly need it, for accurate diagnosis is frequently difficult. We have, however, the inestimable advantage of X rays, if a skilled radiographer is available.

Upper Quadrant Pain.

In the upper quadrant the chief affections will be those of the gall-bladder and of the duodenum.

Gall-Bladder and Duodenum.

Owing to the fact that the gall-bladder may be tucked away beneath adhesions, tenderness is not always elicited, even though it may be decidedly pathological; when present, it is found at a somewhat higher level than that elicited in the presence of a duodenal ulcer.

The type of pain in indigestion is, as indicated previously, usually different, but not uncommonly a barium meal and a Graham's test are needed to differentiate it. Distortion of the duodenal shadow does not always mean duodenal ulcer; it sometimes is due to adhesion and traction by the gall-bladder, as the result of cholecystitis.

Women provide the greater number of cases of gall-bladder disease and men more often have duodenal ulcers. Contributory factors to this are the diet of the one tending to obesity and the excessive cigarette smoking of the other.

The radiation of the pain towards the right subscapular region is much more likely to indicate gall-bladder disease than a duodenal ulcer. Seldom is duodenal pain so acute as that of the gall-bladder. The gall-bladder pain may also be more sudden in its onset and termination, particularly if it is due to a gall-stone.

Although cholecystography has been of immense help, still it is not infallible. I operated upon two patients in one afternoon, in whom the X ray diagnosis exculpated the gall-bladder, yet laparotomy showed the diagnosis to be wrong in both cases. I think sometimes the pain is due to distension of the gall-bladder from kinking of the ducts as a result of ptosis of the liver. A cholecystogram which apparently showed a large gall-stone, misled me for a while, until another skiagram (taken without the dye) showed the shadow to be that of a renal calculus.

Renal Causes.

There is no doubt that renal pain is often referred to the right hypochondriac region, whether it results from stone, hydronephrosis or other causes. The dilated kidney may sometimes be felt in this region; the colon is in front of it, unlike a distended gall-bladder. Of course, a straight-out skiagram may reveal the cause, especially if it is a stone. A good picture will also show the outline of the kidney. Recently the intravenous injection of "Uroselectan B" has aided diagnosis greatly; it not only outlines the shape of the calices and renal pelvis, but gives information as to function. This will prove of great help to the general practitioner, as it may save the necessity of a pyelogram. But up to the present the average excretion urogram is not so good as the average pyelogram. There are also other pitfalls.

Renal calculi are occasional causes of dyspepsias which have resisted treatment by the usual remedies for the more common lesions.

An occasional cause of renal colic is gonorrhœal vesiculitis, due to pressure upon the lower end of the ureter. I have seen several undoubted cases of this. The condition yielded to vesicular massage and the patients remained quite cured. I don't remember having seen mention of this possibility in text books.

Hydatid and Abscess of the Liver.

Hydatid and abscess of the liver must be remembered. The gall-bladder has not the monopoly of pain or swelling in the right upper quadrant.

Cardiac Disease.

Pain of cardio-vascular origin, such as from coronary thrombosis, is occasionally referred to the abdomen, but most usually it is in the epigastric region, rather than to the right side.

Lower Quadrant Pain.

I do not propose to deal with pelvic causes of pain in the right lower quadrant, as they are more in the domain of the gynæcologist.

Appendicitis.

Appendicitis is, of course, the commonest cause of pain in this region. I do not propose to dwell at length on this, except to emphasize what the late J. B. Murphy said, namely, that in almost every case (I think he left out the "almost") the first symptom is pain; vomiting and other symptoms, if present, follow after. Of course, the pain does not always start in the right iliac region; it may be in the epigastric region or on the left side of the abdomen for the first twelve hours.

Pulse rate, temperature and state of the tongue I have found to be very fickle guides, whether in acute or in chronic cases.

Urinary symptoms are sometimes present in appendicitis. I think this is due to a cord reflex. Anatomical position is rarely an explanation.

Gall-bladder trouble not infrequently coexists with appendicitis; the location of the tenderness often leaves us in a quandary; when operating on a gall-bladder, it is my invariable practice to make an oblique paramedian incision that allows me to remove the appendix as well.

I think there I may give a hint that may be valuable to some, and that is, if one has difficulty in exposing the appendix, it is well immediately to enlarge the incision. If, as in my practice, this has been an oblique one through McBurney's point, then prolong it over the rectus muscle, incise the outer part of the anterior rectal sheath, retract the rectus and, if possible, the epigastric vessels, divide the posterior rectal sheath, together with the peritoneum. This immediately gives ample access wherever the appendix may be, whether in the loin or in the pelvis.

Another valuable hint which will sometimes avoid the necessity of enlarging the incision is to do a subserous enucleation. It is particularly useful when the appendix is without a mesentery or when it is bound down by dense adhesions. It is ligatured, divided near its base and buried with a purse-string suture; the serous layer of the appendix is then encircled with an incision and by blunt dissection the appendix is enucleated, just as one would skin a rabbit; at the most, not more than one or two artery forceps are needed.

Iliac Adenitis.

Iliac adenitis (resulting from intertrigo of the groin) is a rare cause of difficulty in diagnosis. I have seen two cases.

Tuberculosis or Actinomycosis.

Of course, the possibilities of tuberculosis or actinomycosis in the ileo-caecal region will not be forgotten. Tuberculosis occasionally affects the Fallopian tube.

Adhesions.

The presence of a scar on any part of the abdominal wall will remind us of the possibilities of adhesions of the bowel or omentum to the parietal peritoneum. This is an important cause of pain. Suspicion will be heightened if there is tenderness over or in the vicinity of the scar, if the pain has no relation to meals, and if it is made worse by exertion, particularly lifting. There are sometimes symptoms of partial obstruction, due to angulation of the bowel. (On adhesions and their treatment I have already contributed an article to The Medical Journal of Australia).

New Growth.

New growth of the caecum or ascending colon will not be forgotten, though it is not so common as in the distal colon. In this latter position, owing to back pressure, pain is not infrequently first felt at the ileo-caecal sphincter.

Visceroptosis.

And now, in conclusion, I shall deal with that most debated of all causes, namely, visceroptosis.

That part of the abdominal contents which is chiefly affected by this condition, is the caecum and ascending colon. In a certain proportion of individuals (females particularly, but by no means only females) the caecum and ascending colon are unduly mobile, being possessed of a mesentery which allows the caecum to drop into the pelvis. The pull on the mesentery, with its contained vessels and sympathetic nerves, causes a stimulation of the latter, with a resultant inhibition of peristalsis and spasm of the ileo-caecal sphincter; this adds to an already preexisting constipation and is a source of pain as the result of the ileo-caecal spasm.

Visceroptosis has further remote effects from the pull upon the right kidney (causing kinking of the ureter and resultant hydronephrosis), upon the gall-bladder and upon the mesentery of the small bowel (with the result that the third part of the duodenum is sometimes pressed upon by the superior mesenteric artery). Urinary and digestive symptoms thus frequently follow.

This ptosis may exist in the long narrow type of abdomen in the young male or female; it may also be present in the female whose abdominal walls have not recovered their tone from frequent child-bearing. The absence of the symptoms in some of the former class is probably due to the contents being kept in position because of intraabdominal pressure being maintained by good abdominal musculature; herein lies a good argument for outdoor games for girls. Although it is held by some that instability of the nervous system, rather than the mobility of the organs, is the cause of the symptoms, I have for many years had considerable doubt of this and have been inclined to view this instability as being partly due to the chronic ill-health which results from the ptosis.

Waugh, Carslaw and Fiddian are British surgeons who have written extensively on this subject; they advocate colopexy and quote very good results. If this is carried out, nephropexy should be unnecessary.

I feel sure this condition is frequently the cause of pain which persists after an appendicectomy. A barium meal will help to diagnose the condition.

THE VALUE OF MEDICAL SERVICES IN RELATION TO PROBLEMS OF DEPOPULATION.[1]

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IN respect of problems of depopulation among a native subject race, medical services are either all-important or worthless. The populations of Melanesia and Polynesia have undoubtedly fallen very greatly during the last hundred years, and the process is continuing. The possibility of the survival or of the extinction of remaining populations is of such vital moment because, if lost, they can only be replaced, if at all, by races bringing with them economic, social and political obligations of a very grave character. The problem of population may be accepted, therefore, as the greatest question in the Pacific so far as Australia is concerned.

The effects of European intrusion upon native races occur in three stages. The first is a period during which the native is attacked by the diseases introduced by the newcomers, by actual arms, by alcohol, by dislocation of tribal life and customs, by over-alienation of land, and by recruitment for labour of the young and virile members of the population. An enormous and immediate decline naturally follows. Practically speaking, this period has long since ended in the Pacific.

The second period is one of partial adjustment, when the European seeks to impose his scale of values upon the native, and the native seeks to imitate European ways of life, in each case with conspicuous lack of success. It is during this period that the native loses the safeguards of savagery and, either through lack of opportunity or ignorance, fails to substitute for them in adequate detail protective European customs.

This condition of unstable equilibrium is seen in many localized areas of Melanesia, in all of which the population is either oscillating about a tentative level or slowly decreasing.

In general terms, one may say that where the native is only rarely visited by white men and continues to devote his energies to the obtaining of his traditional foods in sufficient quantity, he flourishes. Where he comes into contact with Europeans, however, and where, with the prohibition of cannibalism and head-hunting, there disappears also pig-raising and the hunting of the large vitamin-rich fish which are truly regarded by the natives as the "food of warriors", the situation alters immediately for the worse. Where the arrival of planters and traders and the establishment of numerous plantations pin the native down to restricted and often infertile localities, there is a very marked secondary loss of population—a loss which continues and is increased by all those factors which have been described in essays on depopulation.

The outstanding feature of native life in its sociological aspect is that, with partial adjustment to European control, the native becomes resigned to an inevitable decrease in the available food supply, and with the inelasticity that characterizes native habits, makes no attempt, unless assisted, to find new avenues. Unconsciously through the faulty nourishment of the pregnant women, the diminished virility of the offspring and actual increased failure of impregnation, and consciously by a deliberate policy of abortion and infanticide (a custom of the native from time immemorial) he adapts himself to the lowered total of available food stocks. But it is the adaptation of resignation.

So delicate, however, is this condition of unstable equilibrium that very marked effects may be produced upon it by circumstances which would appear otherwise to have only a casual significance.

It is the intensive observation of some one of these factors which, in my opinion, has caused almost every observer who has written on the question of depopulation to emphasize as the sole or a great element some feature really of minor importance.

While medical officer with the British military occupation of German New Guinea during the war, and later as Director of Public Health for that Territory when under mandate, I was able to make a close study of the various factors suggested by different observers. It was soon obvious that actual destruction of native life by violence might be disregarded, and that over-recruitment, though to some extent a factor, was increasingly controlled by government action. The wearing of clothes was found to have no significance one way or the other, where close comparisons could be instituted between populations clothed and unclothed. The patriarchal system of marriage, which had been impugned, was defended by the fact that it has existed from the earliest times, not only through the period of decline, but throughout the whole period of former density of population. It began to be obvious indeed that in a medical sense one must look for, as one would anticipate, factors of a more definite nature to explain depopulation—factors not confined to Melanesia, but universal in their application.

Three phenomena present themselves at once to the medical administrator. They are, first, the heavy infantile mortality rate observable in many parts of Melanesia; secondly, the considerable mortality rate at all ages from preventible diseases; and thirdly, the fact that the great majority of the native populations live on a borderline diet, or alternate between periods of plenty and periods of actual starvation.

An opportunity arose to test these probabilities, and others, in actual practice among the whole population of the Western Islands of the Mandated Territory, and some of you will remember the results, which were published with the Annual Report to the League of Nations of the Territory of New Guinea for 1926-1927.

[1] Read at the twenty-first meeting of the Australian and New Zealand Association for the Advancement of Science, August, 1932.

Until 1923 I had accepted, with some reservation, the psychological factor suggested by W. H. R. Rivers as a primary cause of native depopulation. The actual investigation referred to led me to relegate it to the ranks of effects and complications rather than primary causes. It became only too apparent that the attitude of "hopelessness" upon which such stress had been laid, was itself directly proportional to the degree of local disease prevalence. It arises as a direct sequel of disease, and is not a primary condition resulting from the mere disruption of native customs.

Population was found to be declining in places where native institutions flourished with all their original vigour, as well as where they had been destroyed so long as foodstuffs were scanty and disease rife, and people were found thriving in places where white men had entirely disrupted their social organization (as, indeed, they must inevitably disrupt it everywhere in the course of time), but only where disease was controlled and food abundant.

In Matupi, for example, near Rabaul, the natives have been under white domination for forty years; they have lost almost all semblance of native institutions; they are now a community of native pedlars and hawkers, boat-boys, laundry men *et cetera*, and they are increasing. The secret of this advance is the virtual absence of malaria from that island.

Unia and Garowe are the western and eastern islands respectively of the Witu Group, off the north coast of New Britain. In Unia the population is flourishing; in Garowe it is rapidly approaching extinction. The disease menaces are the same, but Unia has abundant food stocks while Garowe is half starved for staples. If Garowe had as many children born as Unia, and if they survived only to the extent they do at present, the population would be flourishing; if the children of Unia survived only to 50% of the number born, the population would show a heavy surplus which would require and desire employment.

Though, by the courtesy of the League of Nations, my investigations have since been extended from Fiji throughout the whole of the Melanesian groups to the border of Dutch New Guinea, I have seen nothing to suggest any necessity for an alteration of the conclusions at which I arrived, by examination of the declining stocks of New Guinea.

The various permanent agencies found operating might be tabulated as follows: (i) Introduced or accelerated diseases, especially malaria and tuberculosis; (ii) faulty conditions of sanitary environment; (iii) privation and poverty of diet; and (iv) consequential lessened fertility, with frequent miscarriage, deliberate abortion and infanticide.

These causes, again, are merely the two factors of disease and food deficiency, which constitute a vicious circle of decline.

Malaria, tuberculosis, and such chronic diseases produce a mental lassitude and actual bodily weariness which prevent anything but the most essential tilling of the soil and casual foraging for food, while the very poverty of the resultant diet lowers the resistance to those diseases and so provides a vicious circle of more frequent disease and constantly lessened food supplies, until the point is reached at which the human organism can no longer cope with the situation.

The psychological depression found among some native groups is as obviously a consequence of sickness and undernutrition as are the lack of initiative, the lack of vigour, and the despair found among white races in similar circumstances of ill health, poverty and privation.

To suggest that this depression of outlook is a primary cause of depopulation, and to argue (as some have done) that it can only be met by the withdrawal of Europeans and the prohibition of any further intrusion of European influence, good or bad, is to render the situation hopeless indeed and to provide a very ready excuse for doing nothing—an excuse only too readily grasped by the indifferent or the inept.

The attempts made to attack the problem of depopulation are generally as fragmentary and as dissociated as the theories in respect of it. When the matter is discussed at all, the tendency is to introduce local and partial measures, or to try some futile short-cut, based on hope and hearsay. A large body of opinion, it is true, denies liability and expresses the view that annihilation of the native races is natural and inevitable, and that the best solution of the difficulty is to permit the total depopulation of the groups by a policy of masterly inactivity, meanwhile introducing other native labour from abroad.

Another section of popular opinion strongly advocates a mixture of race as a solution of the problem. In the areas to which reference has been made, for example, it was suggested that numbers of Buka Islanders, from the (former) German Solomon Islands, be brought in to "introduce new blood", "because they are more virile", and so forth. The argument for this proposal is utterly fallacious. It amounts merely to the introduction of less diseased natives, so that for a time an outward appearance of health might be given to the population of the group. In the light of this proposal special attention was devoted by me to those foreign natives from Buka, Aitape, Arawe *et cetera*, who were found resident in the groups examined. In no case was there found justification for the statement that they were more fertile, more healthy, or of more value to the community than the average local native. If anything, the indications were to the contrary.

The attitude of the local natives, who are light-skinned Micronesians, had also to be considered. It was obvious that they would bitterly resent the intrusion of any large incursion of what is essentially a foreign race of black Papuo-Melanesians. Verbal assent to the proposal is easy enough to obtain, since the native will agree to anything to please an official questioner, if the question is put emphatically enough. The true opinion of the natives is shown by the fact that of these mixed

marriages, not one foreigner had been given a virgin wife. Most were the widows of local natives, or the discarded women of white men, and their children were locally called half-castes by the natives. The proposal is seen to be antisocial in the extreme.

As to that common fallacy that where a population is weak and unhealthy, a foreign race can revivify it by introducing "new blood", experience teaches that the practice is fatal either to the newcomers or to the original inhabitants. Elsewhere examples have been quoted, and many more could be adduced. The following may suffice to illustrate precisely similar experiments.

Henry Marshall, writing in 1821 of the Africans who composed part of the troops in Ceylon, says that that island "appears to have been extremely unfavourable to the health and propagation of the Caffres. Not a trace of the many thousands brought to it by the Portuguese Colonial Government is to be perceived. The same may be said of a colony of Africans which was imported about the year 1782 by Governor Van de Graaf".

It is interesting to note that these very Africans were imported because the local inhabitants were anæmic, weak and sickly—natural results of the malaria and hookworm diseases that were, and are, rife in Ceylon, and the Africans merely fell victims to those same disorders. Had the Portuguese or the Dutch known how to treat these causes, they would not have needed to lament the lack of stamina of the local Sinhalese.

There is no remedy short of a thorough and comprehensive attack on the causes of the decline, and any scheme for the preservation and resuscitation of island sub-stocks must be a matter of *minutiæ*, where each problem is regarded as an individual project and not as a routine activity which may be assigned to any officer available at the moment. Obviously, too, activities against depopulation will fail utterly in any hands but those of honest and conscientious officers sufficiently interested and enlightened to regard the problem as a life work, for, indeed, it is the problem of a generation. The problem in a medical sense is to combat food deficiencies and the diseases to which they add a fatal virulence.

It had been my intention here to deal in detail with those features of food deficiency which have occupied my attention during the last ten years, but I note that the matter is the subject of a paper by a distinguished colleague, and I can with every confidence leave it to him.

Let it suffice to repeat from my earlier papers that, even at its best, the diet of the Melanesian in his own village is bulky, innutritious, hard to digest, deficient in fat and in protein, and often poor in vitamins *A* and *C*. The deficiencies of the diet are made up by the use from time immemorial of the growing shoots of plants, certain grasses, ferns and fruits, the raw liver of certain animals, and various kinds of fish. To provide a sufficiency demands the unrestricted range of a considerable territory; and since the physical importance of vitamin elements in diet has been dimly recognized by the natives, and, as such, they have often come to figure largely in matters of tribal ritual and taboo, the protection of native institutions which have been built up round them, or the substitution for them of observances equally effective, is important.

Curious sidelights on this matter exist in different parts of Melanesia. The liver of an animal or man is regarded as the seat of all the virtues, and was formerly regarded as the prerogative of chiefs and of warriors in many localities. Certain types of fish, including the shark, all rich in vitamin content, were regarded as of the utmost value and were the staple protein food of the fighting population. In the north-west islands of the Mandated Territory no man could marry until he had captured, single-handed, a shark, and brought it to shore for presentation as food to his intended father-in-law.

The restriction of families with the reduction of food has already been mentioned. In many localities in primitive Melanesia the usual outcome of a diminished food supply is the destruction of infants. In certain localities within very recent times, as, for example, in the Markham Valley of Eastern New Guinea, infants are traded between neighbouring tribes as food.

In more civilized communities, such as those of Fiji, the barbaric aspects of the subject are not seen and date back a hundred years. The population has reached a stage, in the neighbourhood of settlements, where it leases its land to Indian holders and lives upon the rentals. It grows very little native food, except the grosser staples, and supplements these bulky articles more and more, not with essential vitamins, but with tinned foods from the stores.

A curious fact presented to our Health Mission for the League of Nations was the evidence at Vitogo, that the native women reach a stage of infertility at an early age (perhaps after the birth of one child or two) and that this could be and often was corrected by introducing into the diet large quantities of the growing shoots of plants and other items rich in vitamins, which no longer formed a part of their usual diet. This fact is in complete accord with recent investigations on the vitamins associated with fertility.

In the second stage of adaptation to European intrusion, therefore, and if the recent investigations into the importance of foods are correct, it would be probable, considering the present routine diets of the native in his home, to find him poorly developed and short in stature, inclined to the prominent abdomen and thin legs of malnutrition, lacking in energy and initiative, though possessed of occasional considerable endurance, particularly prone to skin ulcerations and such indolent conditions, and liable to succumb very readily to respiratory diseases, especially tuberculosis and pneumonia, and to diseases of the intestinal tract, such as bacillary dysentery. This precisely mirrors the condition that actually is found.

When free natives are brought from their village fastnesses to work under indenture upon plantations or as government labourers, the whole problem of their diet and feeding is rendered much more complicated.

To civilized people such a transfer seems a trivial matter, but to a native it is actually a period of most considerable danger. He has lived in his village from infancy, isolated from the outside world, and because he has been untouched by outside diseases he is surprisingly susceptible to them. Moreover, he has accustomed himself to feeding upon a diet which, though almost always below the optimum scale set by investigators, he has learned to assimilate to the satisfaction at least of his minimal needs.

Whether in his village or on a plantation, then, once this isolation is invaded and its specialized and unstable defences overset by new diseases, a new environment, new conditions of work and new dietetic substitutes, the weaknesses of the native constitution are immediately manifest, and the problem of depopulation is seen in full operation.

You will recollect that I spoke of three periods in the history of depopulation. So far, I have discussed only two—the stage of conflict and destruction, and the stage of partial adaptation and relative equilibrium.

Finally, one of two things occurs. Either the factors for decline predominate and the native race disappears—and this is rare, except in localized communities—or adjustment becomes gradually complete and the native population begins to express this feature in a steady increase.

The end of the first stage may be seen in the New Hebrides; the second stage in all its phases, in our territories in Melanesia; while the beginnings of a successful third stage are seen in Fiji, and are obvious in Samoa, and, of course, in New Zealand.

Apart, however, from the diseases introduced by white men, and deficiences in protein and vitamin-rich stocks available to the natives, even the staple yams and *taro* that make up the main carbohydrate articles of the diet, are threatened. It is generally found that with European plantations come pests that attack these food staples very seriously. For example, in several localities, *taro,* which by native preference and by laboratory experiment is the best of the foodstuffs of the native Melanesian, can no longer be grown on account of the ravages of an introduced pest.

Throughout the whole of Melanesia, apart from the malaria of the north and centre, there are three almost universally prevalent specific diseases, namely, filariasis, frambœsia and hookworm. These and leprosy, which is prevalent to an enormous extent in South Melanesia and to an undetermined extent in North Melanesia, and the tuberculosis and other respiratory infections (which are relatively widespread in the neighbourhood of white settlements, wherever investigation has been thoroughly made, and often far beyond them) with bacillary dysentery, which now appears to be mildly endemic in most parts, and gonorrhœa, which is not only prevalent in areas where labourers have returned from indenture, but is now widely spread, indicate the vast extent of the disease problem in a purely medical sense.

Southern Melanesia, free as it is from malaria, is handicapped to an even greater extent by the immigration of Asiatics and their diseases. The distribution of amœbic dysentery appears to coincide almost exactly with the distribution of the Asiatic population, and the same statement is true with regard to the distribution of the *Ankylostoma duodenale* (the more virulent of the hookworm parasites) and certain other parasitic diseases.

The problem of native administration in a medical sense, therefore (and, indeed, it is increasingly accepted that tropical administration in the final analysis is almost entirely a matter of applied public health), has several phases that go far beyond the range of ordinary medical work. It embraces the activities usually found separated under three heads, the functions of medicine, anthropology and native agriculture.

The value of medical services in relation to depopulation consists essentially in the degree to which medical services are linked with these complementary and cooperative activities. Isolated, they are all relatively worthless; together, they are the basis of progress and, I firmly believe, the only basis. In the past they have been so radically dissociated that effort has been wasted in hackwork and trivial local endeavour.

That phrase "local endeavour", too, provides me with a further theme, and it is my last.

The fault of the medical services of the Administrations of Melanesia has been their discrete and skeleton composition—isolated local services of perhaps two or three medical officers, with provinces as large as mediæval kingdoms to administer and with no uniformity in policy, even amongst their own few officers, no opportunities for research, and no access to information as to the progress made in tropical medicine, even by their neighbours.

What has for many years been the outstanding need of Melanesia is cooperation through a centre for the adequate interchange of medical information, opportunities for a wider field of interest, and better prospects in medical and personal status for the medical officers concerned.

Much has been done by the institution of the Sydney School of Public Health and Tropical Medicine to provide necessary facilities for the education in tropical medicine and hygiene of doctors allocated to tropical services. A necessary uniformity has been attained in quarantine procedure with the Commonwealth of Australia. Something has been done with regard to correlation of information regarding infectious diseases in all administrations in the south Pacific. But there yet remains as an outstanding *desideratum* the matter of uniformity of policy and continuity of programme—the task of a generation. Without it little can be done other than hackwork in respect of native problems of population. With it, and with the cooperation of the anthropologist and the specialist in native agriculture, I am convinced that the third stage of total adaptation, which is the essential for repopulation, can successfully be attained.

Reports of Cases.

A SUPRASELLAR MENINGIOMA.

By A. C. TELFER, M.B., B.S. (Sydney),
Assistant Medical Superintendent, Sydney Hospital.

THE lectures delivered by Professor Dew and Professor Lambie at the meeting of the New South Wales Branch of the British Medical Association have stimulated me to report a case of suprasellar meningioma which came to autopsy at this hospital in 1930.

The case illustrates some of the points made at that meeting. Here, as in so many of these cases, we have an unrecognized chiasmatic syndrome followed by what appeared to be a sudden and fatal increase in intracranial pressure.

The patient was a woman of thirty-five years of age. Unfortunately, little is known accurately of her earlier history. According to friends, she had complained of severe headaches, attacks of vomiting and gradually increasing dimness of vision for the past twelve months. She had seen an eye specialist a few weeks previously, who said she had optic atrophy.

On the morning of her admission to hospital, she fainted, and in the afternoon was discovered in the lavatory in an unconscious condition, with limbs rigid, stertorous breathing and loss of sphincteric control. She was admitted to hospital in this condition. She died about fifteen hours after admission.

Physical examination made was not thorough. The eye and tendon reflexes were present and equal on both sides. The plantar reflex showed a flexor response on the left side and an indefinite response on the right side.

The rest of the examination revealed little of particular value in localizing any lesion.

Post mortem inspection of the cadaver revealed a well nourished female with gross facial features, rather prominent supraorbital margins and lower jaw, high, acutely angled, hard palate. There were no supraclavicular pads of fat, nor were there any abnormal depositions of fat. The whole condition throughout was one of mild acromegaly.

The thoracic organs appeared to be normal, except that there was evidence of old pleurisy at the lower margin at the upper lobe of the left lung, on the axillary surface.

The abdomen presented an unusual appearance, in that practically all the viscera in the upper portion were bound together by adhesions. The liver was particularly involved and pulled out of its normal shape and position by adhesions to stomach and duodenum and transverse colon. The organs were not larger than normal, and their cut surfaces showed no gross irregularity. The pancreas appeared to be normal. The suprarenal bodies appeared to be slightly larger than normal. The left ovary contained a small cyst and the right one seemed to be more fibrotic than normal. The urine contained small amounts of sugar and acetone.

FIGURE I.
View of tumour from above.

When the calvarium was removed, the brain presented a bulging *dura mater*. On opening the meninges it was found that this tenseness was not due to increased fluid, but to a swelling of the gyri, which had proceeded to such a degree that it had obliterated the normal sulcus depressions and had flattened out the usual roundness of the gyri.

On lifting up the frontal lobe, a tumour above the *sella turcica* was discovered. This growth was pressing against and leaving its impression on the cerebral peduncles, the posterior perforated substance and *tuber cinereum*. The infundibulum could not be demonstrated. When the tumour and surrounding bony parts were dissected out, it was evident that it was a suprasellar growth pressing the pituitary gland into the sella and spreading out in a cauliflower-like manner over the margins of the fossa. The antero-posterior and lateral diameters of the tumour measured 3·5 centimetres; it was soft in consistence and had a palish pink tissue-like colour, with an irregular surface. Both optic nerves were pushed laterally immediately at their exit from the optic foramina, and both were flattened out over the tumour, the left one on its lateral surface, the right one on its supero-lateral surface. The appearances of the tumour are shown in Figures I and II.

FIGURE II.
Sagittal section of tumour.

Post mortem radiographic appearances of the feet, hands, pelvis and upper part of the femur seemed to be normal. The blood sugar taken from the superior *vena cava* was 171 milligrammes per 100 cubic centimetres, and the blood did not react to the Wassermann test.

Acknowledgements.

My thanks are due to Dr. Keith Inglis for his aid, also to Miss V. Jones, of the Department of Pathology of Sydney Hospital, for the photographs.

CORONARY OCCLUSION.

By Stratford Sheldon, M.B., Ch.M., B.Sc. (Sydney),
Sydney.

As two papers bearing on the subject of coronary occlusion have recently been published (The Medical Journal of Australia, September 26 and November 7, 1931), perhaps it would be as well to put on record a peculiar case that came under my notice on December 29, 1931.

A labourer, aged fifty-three years, engaged in road repair, suddenly straightened himself and fell backwards. He recovered sufficiently to speak and say he was all right. He was sent to hospital by ambulance, and died within five minutes.

On the margin of the junction between two of the aortic cusps there was a pedunculated polypus consisting of a stalk which was about the diameter of number two catgut, a soft growth, a further portion of stalk, and another small soft growth that was wedged in the opening of the left coronary artery. I have no doubt it was the cause of his sudden death. Neither I nor my colleague, Dr. Palmer, has ever seen a similar condition. The specimen was immediately submitted to the Director (Dr. E. L. Morgan) of the Microbiological Department, the New South Wales Health Department, who reported as follows:

> A small papillary growth, about 1·5 centimetres in length and 0·8 centimetre in width, was attached by a fine stalk to the free edge of the right posterior segment of the aortic valve at the end near the opening of the left coronary artery. In appearance the growth strongly resembled a miniature bunch of grapes, the grape-like nodules being about the size of pins' heads.
>
> Microscopically the general appearance resembles placental tissue. Numerous rounded bodies are present, each consisting of a core edged with a single layer of flattened endothelial cells. The core is for the most part faintly fibrillar, acellular tissue, apparently fibrin. Occasional small strands of fibrous tissue with well formed fibroblasts are present, and these strands often tend to surround areas of fibrin. Some of the papillæ contain scanty minute blood vessels, and scattered here and there are groups of polymorphonuclear leucocytes.
>
> The growth is a fibromyxoma. The possibility of a vegetation was considered, but the fine stalk, the papillary structure and the arrangement of the fibrous tissue all tend to negative this possibility.

As Ewing[a] points out, these tumours are usually covered with blood clot, organized portions of which are fused with the tumour, and are often difficult to distinguish from organized thrombi.

Reference.

[a] J. Ewing: "Neoplastic Diseases", Third Edition, page 188.

Reviews.

BREAST TUMOURS.

The work of Sir George Lenthal Cheatle on tumours of the breast is known the world over. During recent years several of his more important contributions have been discussed in this journal. He has, with Dr. Max Cutler, of Chicago, published a volume entitled "Tumours of the Breast: Their Pathology, Symptoms, Diagnosis and Treatment".[1] We may state at once that this is a book of the utmost value. It has been written in such a way that it will appeal alike to pathologist and surgeon. We cannot imagine that any practising surgeon who has once dipped into its pages will be content until he has a copy of his own and studies it with care and diligence. The pathologist may not agree with all the views expressed (some views conflicting with those of Cheatle and Cutler have been published), but he will feel compelled to treat the work with respect, and will certainly need a copy for reference.

The opening chapters of the book are devoted to the anatomy and physiology of the breast, to the congenital and acquired anomalies of the breast, and to natural laws in pathological growth. In the chapter on natural laws in pathological growths emphasis is laid upon the physiological control which is exercised over the formation of pathological epithelial new growths. The tumours resemble in structure the physiological growth that occurs in the respective glands and tissues in which they grow. The normal function of an epithelial cell of the breast entails its being shed for the purpose of elaborating the secretion of milk. Thus in the breast, as in the sebaceous glands and prostate, cystic states arise which are dependent on the shedding of desquamating epithelium. The breast, thyreoid, prostate and sebaceous glands undergo precisely similar changes of adenomatous tumours, cystic formations and malignant diseases. The chief differences between the pathological changes occurring in them are occasioned by the presence of ducts in the breast and by the desquamative function of its cells. The authors state that there may be no such thing as a benign tumour—"fantastic as the notion may at first appear, all may be malignant and their careers may be interrupted or stopped at any stage". They add that the signs of physiological control in benign and malignant tumours do not lend any support to the view that tumours cannot be due to the introduction of an outside agent. "When only one whole duct of a breast and all its branches are picked out as the site for the growth of epithelial neoplasia, its site is so localized that it looks like the accidental incidence or the selection by design of an outside causative factor." The suggestion of the operation of an outside agent is offered in Paget's disease of the nipple. The clinical signs of Paget's disease are nearly without exception associated with a form of epithelial neoplasia in the underlying breast. The variety of these forms suggests the introduction of an outside agent. The authors conclude this chapter by asking two questions. They ask whether the systemic control over the formation and genesis of benign and malignant tumours is merely one of hereditary tendencies acting upon the affected cells or whether they are influenced by a disturbance in the normal action of internal secretions formed elsewhere. They also ask whether these influences are initiated or influenced by the introduction of an external irritant.

The authors in their classification divide hyperplasia and neoplasia of the breast into: (*a*) those of the epithelium and (*b*) those of the connective tissue. The first group, (*a*), is divided into desquamative hyperplasia and neoplasia (benign and malignant); and the second group, (*b*), is divided into benign and malignant forms. Desquamative epithelial hyperplasia is divided into two forms: mazoplasia and cystiphorous desquamative epithelial hyperplasia. Mazoplasia is the condition commonly known as chronic mastitis. The authors look on the condition as a physiological one and refuse, therefore, to accept the common designation. Cystiphorous desquamative epithelial hyperplasia, on the other hand, is known commonly as chronic cystic mastitis. The authors point out that mazoplasia and cystiphorous desquamative epithelial hyperplasia are in their opinion distinct and separate states; they may each be followed by different events that are highly important in the cystiphorous state, but not so important in mazoplasia. Benign epithelial neoplasia and eventually carcinoma may be superimposed within the actual lesions of the cystiphorus stage, and only occasionally can the formation of fibroadenomata be traced definitely to mazoplasia. "About 20 per cent. of all carcinomata of the breast can be definitely stated to begin within the lesions of the cystiphorous state." All pathologists will not be prepared to adopt this nomenclature.

[1] "Tumours of the Breast: Their Pathology, Symptoms, Diagnosis and Treatment", by Sir G. Lenthal Cheatle, K.C.B., C.V.O., F.R.C.S., and M. Cutler, B.Sc., M.D.; 1931. London: Edward Arnold. Royal 4to., pp. 604, with illustrations. Price: 50s. net.

Admittedly the term chronic mastitis is misleading, but it is open to argument as to whether the description of the authors can be accepted in its entirety; limitations of space prevent further discussion on this point. It should be stated, however, that the authors discuss the clinical aspect of this condition in a way that will be found attractive to most practising surgeons. Particular interest attaches to their description of treatment of mazoplasia by means of ovarian residue.

In discussing carcinoma the authors point out that the lump in the breast, commonly described as the primary tumour, contains secondary deposits as well as the "primary epithelial neoplasia". Primary epithelial neoplasia may have five possible sites of origin and distribution: (i) the nipple, (ii) a localized part of a single duct, (iii) one main duct and its acini, (iv) terminal ducts and their acini, (v) the epithelium of the entire gland or nearly the entire gland. Carcinomata arising from these five sites are discussed in turn. The discussion on treatment is interesting. The authors use radical operation followed by radiation rather than any other method. At the same time, in a separate chapter, they give an excellent account of the radiation treatment of breast tumours.

In the chapter on Paget's disease of the nipple, Cheatle's well known views are given full play. Seventeen case histories are discussed in detail. Stress is laid on the fallacy of treating the surface lesion alone and disregarding the treatment of the underlying breast. The authors agree with Bloodgood that there is only one operation for Paget's disease of the nipple and that is the operation of radical removal.

Enough has been written to show what an excellent book this is. Emphasis should be laid on the clinical discussions that are found throughout. In conclusion, a tribute must be paid to the general make-up of the book and to the beautifully produced illustrations; it would be difficult to surpass them. How so profusely illustrated a book can be published for fifty shillings is difficult to understand.

CONTRACEPTION.

"THE PRACTICE OF CONTRACEPTION" is what it purports to be, "an international symposium and survey" of the subject.[1]

A conference called together by Margaret Sanger, the earliest American worker in this field, was attended by representatives from all the main countries in the world. An opening paper on each method was read, and a fairly full discussion followed; the discussion was probably of more value and interest than the opening addresses themselves.

The greatest contention was caused by Graefenberg's method. This consists of introducing into the uterus spiral rings of gold or silver, varying in size from 1·5 to 3·0 centimetres in diameter. High selection of cases is essential, inflammatory conditions of the pelvis contraindicating its use. The cervical canal needs dilatation up to "number 6 Hegar", when the ring is passed by the special fork introducer. Graefenberg holds that the method is painless and anæsthesia rarely necessary. He admits that bleeding does occur from superficial lesions of the mucosa, and frequently uterine contractions occur, causing pain similar to that of menstruation. It is necessary to watch the patient's temperature for the first three days. A special hook is needed to enable its removal.

The method was adopted by Dr. Norman Haire, who used the ring in 250 cases during fourteen months, rejecting large numbers of unsuitable cases. The objections pointed out by Dr. Haire are: (i) Pregnancy can and does occur in spite of the presence of the ring. (ii) A ring is expelled with or without the patient's knowledge, and, of course, in this case no contraceptive effect can obtain. Notwithstanding these faults, Dr. Haire is continuing to use them.

Leuenbach has had bad results, and in addition he had three patients develop acute inflammatory conditions. The method is neither reliable nor harmless and should not be used.

The statement of the situation in Russia is interesting from the point of view of abortion; enormous numbers of abortions are done without anæsthetic by dilatation and curette, speedily and apparently without bad results.

A paste is used in Copenhagen as an abortifacient, consisting of various ethereal ointments in a soapy basis, that is injected into the uterus and causes abortion in from twenty-four to forty-eight hours.

The contraceptive value of varying chemicals was investigated by Baker, who found that quinine and chinosol, which are often used as a contraceptive, have but slight spermicidal effect, killing only at a concentration of 0·5%. Soap has a high spermicidal value, sodium oleate killing spermatozoa at a concentration of one in 132. Lactic acid kills sperms at a concentration of one-sixteenth *per centum*, and is used in jellies containing 1% to 2%.

The wish bone or collar stud type of pessary was unfavourably commented upon, and virtually none of the clinics used it.

It was generally agreed that the premenstrual week can be considered as at least a relatively safe week.

Knaus goes further and states that conception can occur only from the eleventh to the seventeenth day of the cycle, but this was disputed.

The contraceptive method most commonly employed by the varying clinics is the vaginal diaphragm, generally combined with spermaticidal jelly or ointment. Varying opinions are expressed as to size and the material of the ring expanding the diaphragm.

Cervical caps are recommended in some cases, and it is universally agreed that each individual should be fitted by a competent clinician, and one, too, who has put some thought and study into the matter.

That there is a strong demand for more precise knowledge of the subject is made evident by the numbers of investigating clinics and the number of recent publications.

HYDROTHERAPY.

THE skin is one of the most important organs of the body. Its wide extent, the vast ramifications of capillaries in it, its sensitiveness to external impressions, and its excretory function make of it an excellent medium through which certain forms of treatment may be administered. In "Hydrotherapy and Physiotherapy for Bath Attendants, Nurses and Biophysical Assistants" Dr. Lionel C. E. Calthrop deals with treatment of disease through the skin by means of water, and more briefly of physiotherapy.[1] The book is intended chiefly for bath attendants, as its title implies, but it is of interest to others as showing the many ways in which water, and other substances, such as mustard, peat, various forms of fango or mud, and paraffin, may be applied and the changes they are capable of effecting. It is questionable whether sufficient use is made of these remedies, which, if properly administered, are rarely a source of harm, and, from their direct effects on the skin and indirect action on more distant parts, are of undoubted use in some conditions of ill-health. The book is written in a clear and understandable manner; it has several illustrations, and it should be of use to those for whom it is intended, and to others who may find interest in the study of hydrotherapy. Physical therapy is dealt with briefly, yet sufficiently to assist those for whom the author writes.

[1] "The Practice of Contraception: An International Symposium and Survey", edited by M. Sanger and H. M. Stone, M.D., with foreword by R. L. Dickinson, M.D.; 1931. London: Baillière, Tindall and Cox. Royal 8vo., pp. 334, with 22 illustrations. Price: 21s. net.

[1] "Hydrotherapy and Physiotherapy for Bath Attendants, Nurses and Biophysical Assistants", by L. C. E. Calthrop, M.B., M.R.C.S., L.R.C.P.; 1931. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 184, with illustrations. Price: 5s. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 15, 1932.

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MATERNAL MORTALITY AND MORBIDITY.

In June, 1928, a special Committee was commissioned in Great Britain by Mr. Neville Chamberlain "to advise upon the application to maternal mortality and morbidity of the medical and surgical knowledge at present available, and to inquire into the needs and direction of further research work". Sir George Newman, of the Ministry of Health, was appointed chairman, and the members of the Committee were: Professor F. J. Browne, Dame Janet M. Campbell, Dr. Ethel Cassie, Dr. L. Colebrook, Professor A. Donald, Dr. C. E. S. Flemming, Sir W. M. Fletcher, Dr. H. Kerr, Dr. W. H. F. Oxley, Professor M. H. Phillips, Dr. C. E. Tangye, Professor O. L. V. S. de Wesselow; Dr. Jane H. Turnbull was appointed secretary. An *interim* report was issued in 1930 and the final report has just been published. The report is a document of 157 pages and is full of most important information; we would that it could be studied by every medical practitioner in the Commonwealth. In the present instance it will be possible to direct attention only to the general scope of the inquiry and to the more important conclusions. Other aspects will be discussed on a future occasion.

The number of maternal deaths covered by the whole report was 5,805 (2,000 in the *interim* report and the remainder in the final report). Of the 5,805 maternal deaths, 4,655 were directly due to child-bearing and 1,150 were the result of intercurrent disease. The cause of death in the 4,655 cases was as follows: sepsis, 13·1%; eclampsia, 11·6%; operative shock *et cetera*, 9·9%; *ante partum* hæmorrhage, 8·0%; *post partum* hæmorrhage, 6·3%; other toxæmias, including chorea and mania, 6·0%; embolism, 6·8%; abortion, 12·4%; extrauterine gestation, 1·6%. It was found that more than one factor was often concerned in the death, but in many instances a definite defect in maternal care was clearly the starting point in the patient's downward course. This defect was called by the Committee the "primary avoidable factor". Of the 4,655 cases in which death occurred, 1,576 had an obvious primary avoidable factor; in 1,856 cases no departure from established practice, having a causal relationship to the death, was found; 653 cases were not strictly relevant to this part of the investigation (death was due to abortion or to extrauterine gestation); in 570 instances the information available was insufficient to justify the drawing of conclusions. The Committee points out that if the cases not relevant to this portion of the inquiry and those in which the information was not considered sufficient to warrant a definite conclusion are deducted, the percentage of deaths in which a primary avoidable factor was evident was 45·9. All students of the subject will be prepared to admit that maternal death in obstetrics will never be entirely eliminated—in a certain number of cases death will be unavoidable. At the same time it is safe to conclude that the percentage in which a primary avoidable factor was operative was higher than 45·9; errors in technique would not all be recorded, nor would every error in judgement be obvious in the reports supplied to the Committee. Even if the percentage 45·9 be accepted as correct, the conclusion that at least one-half of the maternal deaths occurring in Great Britain are preventible is a standing reproach. There is no reason to suppose that an investigation carried out in Australia would yield results differing greatly from those obtained in Great Britain. Ante-natal care in Australia is not always complete, the judgement of midwife and medical practitioner in Australia is

not invariably sound, nor is their technique always flawless; all Australian patients are not trained to make full preparation for confinement, nor are Australian facilities for treatment all that might be desired.

The choice of the term primary avoidable factor was a happy one. Obviously, if a causative factor in a death is avoidable, it should be avoided. The members of the Departmental Committee are convinced that the primary essential for the reduction of a high maternal mortality is sound midwifery, before, during and after child-birth. They add naïvely that this does not depend chiefly upon administrative arrangements or the expenditure of public money. Sound midwifery has been discussed almost *ad nauseam* in this and every other medical journal read by general practitioners and obstetricians. The Departmental Committee describes imperfections in the management of pregnancy and labour and imperfections of maternity service. The imperfections in management are discussed under the headings of sepsis, toxæmias, hæmorrhage and preexisting disease. Imperfections in maternity service are held to exist in regard to training of students, facilities for post-graduate instruction, specialization, midwives, ante-natal supervision and hospital accommodation. It is quite unnecessary to discuss in this place the principles of sound midwifery practice. Soundness of midwifery practice depends almost entirely on the will of the obstetrician to carry out what he knows to be the correct procedures. The word procedure in this connexion includes not only the actual attendance at delivery, but also ante-natal supervision and the instruction of patients, whenever possible, in the need for preparation for pregnancy and for general supervision of health. The voluntary checking of midwifery records and correction of errors can best be carried out in the way adopted by the Obstetrical Committee of the Queensland Branch of the British Medical Association. (The work of the Queensland Branch was discussed in this journal on January 30, 1932.) In several States in Australia post-graduate courses in obstetrics are held, but not in all. Efforts should be made to hold such courses regularly and in all the States. At post-graduate courses in obstetrics attention should be paid to the normal course of labour as much as to abnormalities and complications. It is the underlying principles and rudiments of the art that require revision. Of specialization much might be written. Too little attention is paid to it. There is not much encouragement for any medical practitioner to perfect himself in the difficulties and intricacies of obstetrics. In these days, when more hospital beds are being allotted to obstetric patients, there is more opportunity for specialization; and when a practitioner is known to have acquired more than usual proficiency as an obstetrician, his services should be sought by his brother practitioners in times of difficulty.

Current Comment.

HYPERNEPHROMA.

Considerable confusion still exists regarding the use of the term hypernephroma. One school of thought looks upon hypernephromata as arising from the kidney and another holds that they have a suprarenal origin. There was justification for the view expressed by Derrick, of Melbourne, in this journal about ten years ago, that the use of the term hypernephroma should be abandoned. The name, however, still remains, and with the uncertainty of the origin of these tumours there is doubtless in the minds of many no clear conception of their clinical picture. J. Gottesman, D. Perla and J. Elson have published the results of a study of forty-four cases of hypernephroma, in thirty-three of which autopsies were obtained.[1] They describe a hypernephroma as a neoplasm of the kidney or suprarenal gland which is yellow and vascular, which shows a tendency towards hæmorrhage, necrosis and cyst formation, and which microscopically resembles in some areas the structure of the suprarenal cortex.

In twenty-eight instances in the series of forty-four, clinical manifestations of neoplasm were present; in the remaining sixteen the hypernephroma was discovered accidentally at autopsy. The ages of the patients varied from thirty-five to seventy years; the greater number of tumours occurred in the fifth and sixth decades. The most frequent mode of onset was with urinary symptoms, such as frequency of micturition, tenesmus, nocturia, inability to pass urine, and hæmaturia. Sometimes abdominal pain or pain in the lumbar region was the initial complaint. In some instances no symptoms were noted until a mass was accidentally discovered during the course of an examination. In a few instances loss of weight and weakness were the

[1] *Archives of Surgery*, May, 1932.

first abnormalities noted. The duration of the illness from the manifestation of the first symptom until death occurred ranged from six months to twenty-two years, the average period being four years and four months. In about half the cases the primary symptoms were referable to metastases.

Gottesman and his two collaborators point out that all types of histological variations may occur in the same tumour, variations such as a cortical adenoma, a typical hypernephroma, papillary and adenomatous forms and highly malignant carcinoma and sarcoma-like infiltrations. They add that the almost constant occurrence of papillary structure in some areas of the hypernephroma makes any arbitrary classification on the basis of morphological variations useless and unnecessary. This statement is open to argument. It is certain, however, that whatever classification is adopted, there will always remain a certain number of tumours of this region which will be labelled with great difficulty. We do not intend to make further reference to the question of classification. Rather would we emphasize the clinical features of hypernephromata. The variety of symptoms associated with them and the occasional absence of symptoms are remarkable. Since metastases are sometimes the first abnormalities noticed, the possibility of a tumour of the kidney being responsible for them must be borne in mind. Gottesman and his fellow workers think that when metastases have been present for years, the metastases occurred at a stage of relatively low malignancy of the primary tumour. They draw an analogy between these relatively benign metastases and the so-called benign metastases sometimes associated with carcinomata of the thyreoid. These thyreoid tumours have been discussed in these pages on previous occasions. These secondary deposits obviously cannot be regarded as benign. On reading Gottesman's article one cannot help thinking of the statement of Cheatle and Cutler, in their book on tumours of the breast, that, fantastic as the idea may appear, there may be no such thing as a benign tumour: "all may be malignant and their careers may be interrupted or stopped at any stage". Why and how this happens is the ultimate problem of malignant disease.

INFLUENZAL MENINGITIS.

AMONGST the various microorganisms responsible for causing an acute meningitis, the *Bacillus influenzæ* is recognized as an occasional delinquent. But although influenzal meningitis is distinctly uncommon, since it cannot be identified without adequate laboratory services, it is possible that its incidence is greater than is thought. A point of peculiar interest about this condition is that it is regarded by many as being inevitably fatal, and, indeed, this view has been given currency in some text books. But serious though this disease is, it does not always strike to kill, as local experience has shown. A. P. Hart has reported a personal series of 32 cases observed in the last eight years at the Hospital for Sick Children at Toronto; among these two patients recovered.[1] It is of interest that only seven out of the thirty-two patients were recognized as suffering from a meningitis when admitted to hospital. In a review of the symptomatology Hart points out that there is nothing distinctive about the disease. Vomiting was common; drowsiness, fever and irritability also frequently occurred at the onset. Convulsions were described in less than half the cases, and this was never an initial symptom. A very interesting observation was made concerning the respiration rate; this was sufficiently rapid in many instances to suggest a diagnosis of pneumonia. Most of the children were rigid in the neck and often had actual head retraction, and were so noticeably tender on handling that the author thinks this sign is of considerable frequency and importance. The leucocyte count was usually over 10,000 per cubic millimetre, but in a few cases was not elevated above the normal. The cerebro-spinal fluid cell count was invariably high, 500 to 4,000 or more.

Inquiry was made with a view of ascertaining if the meningitis was secondary to an influenzal infection of the respiratory tract. So far as could be traced, and the point is difficult to prove where the patients, like these, are young children, the disease was almost always primary. It is worth noting that the *Bacillus influenzæ* was grown from the blood of several of the patients. In view of this, and remembering the analogy of the meningococcal infections, it is not surprising that arthritis occurred several times as a complication.

The patients who recovered were treated by repeated lumbar puncture and the judicious use of glucose saline solution. In addition, transfusion of blood was employed, in conjunction with exsanguination venesection in one case, and in the other with the intramuscular injection of blood serum from a supposed convalescent. Hart points out the well recognized difficulty in treating a disease like meningitis, that of applying any treatment likely to be effective at a sufficiently early stage in the illness. Too often the first fluid withdrawn from the spinal theca is already purulent. However, one child who recovered was only three years of age, and this gives encouragement for the early institution of some active line of treatment. The empirical method of attempting to supply the patient with antibodies by introducing either whole blood or serum from a healthy donor can be employed, or a definite effort made to use a specific method by obtaining convalescent serum for use. One real difficulty here is that our knowledge concerning the ætiological relationship of the *Bacillus influenzæ* to the disease known as influenza (itself at times a rather vague entity) is not very precise, and it may be difficult to know just who is a convalescent. But where epidemic conditions, even if they are very local, exist, contacts who are convalescent from a recent febrile illness might well be utilized as donors of blood or serum.

[1] *The Canadian Medical Association Journal*, August, 1932.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Heredity of the Agglutinogens M and N of Landsteiner and Levine.

ALEXANDER S. WEINER, SIDNEY ROTHBERG AND S. A. FOX (*Journal of Immunology*, July, 1932) present data to support the medico-legal application of the agglutinogens M and N of Landsteiner and Levine. According to the theory of Landsteiner and Levine, the inheritance of the agglutinogens M and N depends upon a single pair of allelomorphic genes, M and N. Accordingly there are only three genotypes possible, MM, MN and NN, corresponding to the plenotypes M+N–, M+N+, and M–N+, so that bloods lacking both agglutinogens are impossible. In ten thousand individuals examined thus far not a single such blood has been found. It is therefore a simple matter to determine what the types of the children must be when the types of the parents are known, and a table setting out the percentage of children of each type is presented. For medico-legal purposes the mechanism of heredity of the agglutinogens M and N may be summarized as follows: (i) The agglutinogen M cannot appear in the blood cells of a child unless present in the blood cells of one or both parents. The same law applies to the agglutinogen N. (ii) The combinations M+N– parent with M–N+ child, and M–N+ parent with M+N– child are impossible. If the theory of Landsteiner and Levine is correct, an exception to either of these two laws must be due to non-paternity. For exclusion by the first law the types of both parents must be known, for the exclusion by the second law the type of only the parent in question is necessary. A critical test of the theory can be made by the examination of a large series of mothers and their children to determine whether or not combinations of M+N– mothers with M–N+ children, and M–N+ mothers with M+N– children exist. By considering the mothers only, the possibility of illegitimacy is excluded. In the authors' series four hundred and sixty-one mothers and four hundred and ninety-seven children were examined without a single critical combination being discovered. The exceptions to the rule are only reasonably explained by illegitimacy. Full details of the technique employed in the tests are given and a word of warning is issued that the tests in medico-legal cases should be carried out only by individuals who have done a considerable amount of work on the agglutinogens M and N. The medico-legal applications of the tests may be used in the following ways: (i) For the determination of non-paternity. Where neither parent has the agglutinogen M (or N) but the child has that property, paternity is disproved. Furthermore, if the putative father is of type M+N– and the child of type M–N+, or *vice versa*, non-paternity is proved. Where the man accused of paternity is not the true father, he can be exonerated by means of the agglutinogens M or N in 18·75% of cases. Fully one-third of innocent men can be cleared by the combined use of the agglutinogens A, B, M and N. (ii) For detecting interchange of new-born infants in hospitals. For the distribution of groups present in New York City 42·42% of these cases can be solved by examining the bloods of the four parents and the two infants for the agglutinogens M and N. The chances of detecting interchange of infants by means of all four agglutinogens, A, B, M and N, are approximately two out of three. (iii) For determination of non-maternity. If the supposed mother is of type M+N– and the child of M–N+, or *vice versa*, non-maternity is proved. Where it exists it can be detected in 12·5% of the cases by means of the agglutinogens M and N. The chances of proving non-maternity by means of the Landsteiner blood groups is less than 5%.

Poliomyelitis.

BEATRICE F. HOWITT (*Journal of Infectious Diseases*, January, 1932) summarizes the results of an investigation to determine the effect of intramuscular treatment with sera on monkeys infected with poliomyelitis as follows. Seventy-nine monkeys were used in five different experiments, in two of which the virus was injected intracranially and in three intranasally. Of fourteen animals treated intramuscularly during the preparalytic stage an average of 81·6% recovered, although all except one had some form of residual paralysis. Of the eleven treated in the same stage of the disease by the combined intrathecal and intravenous method, an average of 33·3% recovered, though not without paralysis. In the group of fourteen untreated controls, 54% recovered with varying degrees of paralysis. There was 100% mortality when treatment was given by either method in fourteen animals showing early or advanced paralysis. Seventeen of the thirty-seven animals given intranasal instillations of virus did not acquire the disease, even though four of them received six different instillations. These animals were not immune, as they later succumbed to intracranial injections of virus. The disease produced by the intranasal route in monkeys is in every way comparable to that produced by the intracranial, except that the period of incubation may be prolonged. The prophylactic use of the serum of monkeys convalescent from poliomyelitis was effective when given intramuscularly either twenty-four hours before or twenty-four hours after the intracranial injections of virus, but was apparently without effect when given simultaneously with the virus. Treatment by the combined intraspinal and intravenous method at the same interval before or after intracranial inoculation proved entirely ineffectual. As far as can be judged, the intramuscular use of serum from convalescent monkeys was as effective as, if not more effective than, the combined intrathecal and intravenous method. In the small series in which repeated doses of serum were given the results seemed no more effective than after one dose. If the serum is administered during the prodromal or preparalytic stage, the prognosis is more favourable than if it is given later, as paralysis or even death was never prevented when treatment was delayed until the advanced stages.

BEATRICE F. HOWITT (*Journal of Infectious Diseases*, January, 1932) reports the results of prolonged inoculation of two goats and one sheep with the virus of poliomyelitis and shows that antiviral properties may be developed in these animals. The goats and sheep used were shown by numerous tests not to contain natural antibodies in their serum. The potency of the serum produced in all three animals was increased after the intracutaneous and subcutaneous methods of inoculation were added to the intravenous. The immune sheep serum was found to contain antiviral bodies of higher potency than those reported by Fairbrother in immunized horses, and the sera of both goats compared favourably with that of the horse. Prolonged immunization of the two goats and the sheep was necessary. The antiviral substances produced were found to be capable of protecting monkeys against infection.

Bacteriæmia and Changes in Temperature.

HARRY WEISS AND REUBEN OTTENBERG (*Journal of Infectious Diseases*, January, 1932) report the result of an investigation into the relationship between bacteriæmia and changes in temperature. Four cases of subacute bacterial endocarditis were studied, and the results obtained indicate that a relationship between changes in bacteriæmia and fluctuations in temperature probably exists. It appears that a rise and fall in the bacteriæmia are followed by corresponding changes in temperature. The results also indicate that the bacteriæmia is maintained by at least two factors, a constant, relatively uniform washing into the blood stream of bacteria from endocardial vegetations, and a sudden liberation of a large number of organisms into the circulation, producing the phenomenon of so-called "bacterial shower". An interesting feature of the bacteriæmia is its relative constancy in any one infection, the rate of removal corresponding with remarkable accuracy to the rate of invasion. It was noted that the clinical course of the case bore no fixed relation to the degree of bacteriæmia. The occasional showers are not followed by proportionate changes in the temperature or in the symptoms. Infections with

low bacterial counts may have a severe clinical course and come to a fatal termination early, while other infections with much higher counts may pursue comparatively mild courses over much longer periods. The results indicate that if diagnostic blood cultures are taken just before the expected rise of temperature, instead of at the time of hyperpyrexia, more positive results will be obtained.

HYGIENE.

The Organization of Treatment for Industrial Rheumatism.

A. R. NELIGAN (*Journal of State Medicine*, May, 1932) states that on the Continent facilities for treatment of industrial rheumatism are looked upon as a matter of public health and arrangements for treatment fall under four heads: (i) spas, (ii) physical treatment departments of general hospitals, (iii) special hospitals, (iv) town clinics. He refers to examples of such organizations. The Medical School at Strasburg has a chair of medical hydrology and associated laboratories. At Brussels there is a "rheumatic centre" at the great General Hospital Brugman. This is in charge of the professor of physical medicine at the university. He gives details of work in Sweden. The tendency in the United States of America is to organize special departments at both the general and special hospitals, called "arthritis clinics". Adequate facilities in Great Britain fall very short of what are required. The problem should be recognized as a matter of public health as well as of economics, and physical methods should be regarded as usually necessary at some stage. Such physical treatment can be properly given only in an institution. Education of all concerned, including the medical profession, is essential for practical success. Many of the public are losing confidence in the ability of the general practitioner to deal with the problem of chronic arthritis and are therefore consulting quacks and taking nostrums. Propaganda, both by public health services and private organizations, is necessary. Prevention is of first importance, and here housing reform, playing fields, working conditions, avoidance of strain, selection of workers for different tasks, provision of pit-head baths, and efficient treatment of local sepsis, especially of the teeth and throat, must be considered. In considering the organization of treatment he asks the two questions: (i) If institutions for physical treatment are necessary, how and where are they to be provided? (ii) Will the results justify the expense, admittedly considerable? Spas get the patient right away from home and work into favourable surroundings and suitable *régime*. Hospitals should be reserved for bed patients and hostels provided for the majority who are ambulant. Four types of institutions are desirable: (i) An arthritis unit adjacent to and in cooperation with a large teaching school of medicine or a university. (ii) A rheumatism department of a hospital, whether general or orthopædic. (iii) Special clinics, the most popular way of tackling the problem. They have the advantages of a specially chosen staff concentrating on a single end, and can readily cooperate with existing institutions, thus avoiding a laboratory and equipment. (iv) Hospitals for chronic disease, of which rheumatism is the commonest. Intensive investigation and treatment are necessary if serious incapacity is to be avoided. Therefore, some beds should be set aside for the investigation and treatment of chronic disease.

The Diagnosis of Hookworm Disease.

GEORGE W. BACHMAN AND R. RODRIQUEZ-MOLINA (*The American Journal of Tropical Medicine*, July, 1932) report the results of their investigation into the effects of the intradermal injections of extracts of various helminths and *Bacillus coli*. They made extracts of *Necator americanus*, *Ascaris lumbricoides*, *Fasciola hepatica* and *Bacillus coli*, and tested large numbers of persons infested with intestinal worms, and large numbers of apparently healthy persons. They report that certain individuals possess a pronounced hypersensitivity to extract of *Necator americanus*, but that in a large number of instances there is a similar sensitivity to extract of ascaris. The authors conclude that the application of intradermal tests in the diagnosis of infestation with *Necator americanus* is of no practical value.

Health of Workers in the Engineering Industry.

ARTHUR MASSEY (*Journal of State Medicine*, October, 1931) discusses the health of engineering workers in Coventry from the occupational and the community aspects. Pointing out that the health of the workers depends in some measure on the prosperity of the industry, the personal and environmental factors of industrial health are contrasted. The health of the individual in the first place will influence to some extent his choice of occupation. Engineering is on that account preeminently a male vocation, and physical misfits are naturally eliminated at an early age by selective transfer to other trades. The mortality is relatively low, but the morbidity is not necessarily so, on account of the high proportion of young unmarried men in this industry. Environmental factors relate to the suppression of dust, structural conditions and cleanliness, the provision of ventilation, sanitary accommodation and recreational facilities. Among occupational factors certain hazardous processes in engineering are discussed. Of these sandblasting needs stringent regulation, and shotblasting should be considered as an alternative. The processes of chromium plating and cellulose spraying can be controlled to produce no ill effects by provision of suitable protective clothing and exhaust ventilation. The problem of boils and dermatitis from oil appears to have been solved by the addition of a suitable bactericide to the oil. The personal factor is associated with 75% of accidents, and scientific selection of employees is a fundamental measure in their prevention. Education in safety, preservation of general health, and protective devices are other factors in order of importance. Fatigue is largely governed by the general health of the worker and the working environment. The use of modern tools and avoidance of monotony should be aimed at in preventing fatigue.

The Ætiology of Rheumatism.

WILLIAM WILLCOX (*Journal of State Medicine*, April, 1932) discusses the ætiology of rheumatism. After excluding pain and inflammation in joints, muscles and fibrous tissues, caused by well known specific diseases, gout, injury, and central nervous diseases (Charcot's disease *et cetera*), together with hæmophilia, scurvy and "serum fever" arthritis, he divides rheumatism into acute rheumatism or rheumatic fever and chronic rheumatism, which includes various forms of arthritis and fibrositis. He supports Sir George Newman's description of rheumatic fever as the rheumatism of childhood, and chronic joint rheumatism as the rheumatism of the adult, and he draws attention to the organic changes in the joint, often with bony deposits, which occur in chronic rheumatism. Rheumatism is due to streptococci, different forms being present in the two types. Rheumatism commonly attacks children of the working classes, though not necessarily the most poverty-stricken children. It is a disease of environment, damp housing and the damp months of the year being associated, basement dwellings especially so. Reduced resistance, due to absence of sunlight, overcrowding and diet deficiency, favours the development of rheumatic fever. Protein, fats and the necessary vitamins protect against disease. Vining has shown that the absence of vitamin *D* favours the development of dyspeptic conditions, the common precursor of rheumatic fever. Among children of the better-to-do rheumatic fever is comparatively rare, yet amongst the poorest of the poor rheumatic fever is not more common than in the prosperous artisan class. Unhealthy large tonsils harbour streptococci—a very common form of infection. Though rheumatic fever is due to a special streptococcus, it does not appear to be contagious. The author has never seen the disease transmitted in hospital from one person to another. Isolation is unnecessary. The suitable soil and the environment are required as well as the seed.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Melbourne Hospital on May 18, 1932. The meeting took the form of a series of clinical demonstrations by the members of the honorary staff. The first part of this report was published in the issue of October 8, 1932.

Injury to Spinal Cord.

DR. H. F. MAUDSLEY showed a male patient, aged thirty years, who, in a motor accident two years before, injured his cervical cord. The left arm was paralysed, with hyperæsthesia over the cutaneous distribution of the fourth and fifth cervical segments. There was also loss of sensation to heat and cold, with presence of sensation to light touch over the eighth cervical and first and second thoracic segments. A diagnosis of hæmatomyelia was arrived at.

X ray examination of the cervical and upper thoracic vertebræ revealed fracture of the seventh cervical transverse process with non-union, as well as a fracture of the transverse process of the first dorsal vertebra.

Dr. Maudsley pointed out that there was a residual paresis of the triceps, deltoid and biceps muscles, and there was some paresis of the muscles of the forearm, together with wasting of the affected muscles. There were no objective sensory findings in the region of the upper arm and forearm. There was loss of all forms of sensation over the palmar surface of the hand and the dorsal surface of the fingers. The patient complained of severe neuralgic pains over the anæsthetic area. This pain was becoming progressively worse and prevented any use of the affected hand, on account of the extreme pain that he suffered when he attempted to grasp an object.

The question arose as to whether any operative procedure over the affected portion of the cord would give the patient any relief from his pain. The pain did not appear to be a root pain, therefore it was not due to pressure exerted by the fractured transverse processes. In all probability pain was due to irritation of the central crossing pain fibres in the injured portion of the cord. These fibres were paralysed so far as conduction of all forms of sensation from the affected hand was concerned, but irritative changes in the region, possibly of a regenerative nature, gave rise to the localized pain. If this was a correct interpretation of the phenomena described, operation was unlikely to give the patient relief from pain, and after such a time had elapsed since the accident the arm paresis could scarcely benefit.

Subacute Bacterial Endocarditis with Coronary Thrombosis.

DR. MARION WANLISS, in the absence of Dr. Douglas Thomas, and on his behalf, showed a returned soldier, aged thirty-six years, a married man, who was admitted to hospital on June 12, 1931, complaining of severe substernal pain, lasting for some hours, associated with dyspnœa, restlessness, shivering and sweating. Some dyspnœa had been present for ten years, increasing in severity during the past three weeks. The past history was one of osteomyelitis of the left tibia at the age of six years, the scar from which broke down again after a wound sustained in 1916 and which discharged until two years ago. There was also a history of growing pains while a child. Six years previously the patient had been in hospital for six months and for the same period four years previously with rheumatic fever.

On examination on June 12, 1931, the patient's temperature was 37·8° C. (100° F.), his pulse rate was 84 and his respiratory rate 26. His systolic blood pressure was 120 and his diastolic pressure 50 millimetres of mercury. The apex beat was situated in the sixth intercostal space 13·75 centimetres (five and a half inches) from the midline. There was one finger breadth of right cardiac dulness. Generalized diastolic and systolic murmurs, maximum at the base, were audible; and the apical systolic murmur was conducted to the axilla. The aortic regurgitation was so marked that capillary pulsation was noticeable in his cheeks. Moist sounds were audible at the bases of the lungs. The abdomen was tender in the splenic area, with the spleen just palpable. No abnormality was detected in the central nervous system. The specific gravity of the urine was 1·022, it contained albumin and pus cells at the time of the patient's admission to hospital, and red blood corpuscles were found on several occasions. The leucocytes numbered 11,000 per cubic millimetre. Blood culture yielded non-hæmolytic streptococci. The blood urea measured 48 milligrammes per hundred cubic centimetres. The Wassermann test applied to the blood gave a negative result. The electrocardiograph was very suggestive of coronary thrombosis. During the patient's stay at the hospital, he developed a friction rub at the base of the left lung and some petechial spots on the thighs.

On May 10, 1932, the patient's only complaints were dyspnœa and occasional œdema of the ankles. He was apyrexic at morning and at night. His pulse rate was 72, the systolic blood pressure was 150 millimetres of mercury and the diastolic pressure 80. His colour was good and there were no petechiæ; there were slight clubbing of the fingers and marked capillary pulsation. There was mastitis of the right breast. The cardiac apex beat was in the sixth intercostal space, 12·5 centimetres (five inches) from the mid-line. An apical systolic murmur was conducted into the axilla and generalized systolic and diastolic murmurs were maximum at the base. The vocal resonance at the bases of both lungs was diminished; no adventitious sounds were heard. On examination the abdomen was found to be tender in both loins and also in the splenic area. No rub was audible and no spleen was palpable. No abnormality was detected on examination of the fundi and there were no petechiæ on the skin or conjunctivæ. There was no œdema of the feet or sacrum. The specific gravity of the urine was 1·012, it was acid and contained albumin; no red blood corpuscles or casts were detected in the urine. The urea concentration test yielded a figure of 2%. The leucocytes numbered 13,800 and the red blood corpuscles 5,650,000 per cubic millimetre, the hæmoglobin value was 85% and the colour index was 0·82. No growth appeared on incubation of the blood. The electrocardiograph still showed a coronary type of curve with occasional ventricular extrasystoles. The blood sedimentation test showed an abnormal curve of "quiescent" type with a sedimentation index of 22 millimetres.

The discussion centred mainly round the possibility of the osteomyelitis being the origin of a non-hæmolytic streptococcal endocarditis, the occurrence of the coronary thrombosis probably due to embolism just before the patient's admission to hospital, also his surprisingly good general condition, despite signs of activity as evinced by the leucocytosis and blood sedimentation curve.

Obscure Pulmonary Condition.

Dr. Wanliss next showed a male patient, a Dane, aged twenty-eight years and single; his health had been excellent until fourteen years previously, when he contracted influenza. Two months after this he had a hæmoptysis after severe exertion; his sputum at this time yielded no growth on attempted culture. Since then he had had repeated small hæmoptyses, perhaps twice weekly, and with rarely more than a fortnight's interval between them. His general condition had been excellent and he was in the amateur boxing ring two years ago. He complained not only of fatiguability but of coughing up about half a cupful or less of sputum *per diem*, offensive to taste and smell. On examination the patient was seen to be a young man with a magnificient physique, weighing eighty-one kilograms (twelve stone twelve pounds). His fingers were very markedly clubbed. His temperature was 36·7° C. (98° F.), his pulse rate 66, the respirations 24 per minute, the systolic blood pressure 120 millimetres of mercury and the diastolic pressure 80. Physical examination revealed nothing abnormal. X ray examination with a screen revealed moderately good diaphragmatic excursion and a dull area laterally and posteriorly at the base of the upper lobe of the right lung. The film showed infiltration throughout the first and second interspace

areas of the apex of the right lobe, indicating chronic tuberculosis. No definite bronchiectatic changes were seen, though there were several areas in the lower lobe of the right lung suggesting bronchial dilatation.

When lipiodol was used, it was found that there was in the apex of the lower lobe (corresponding to the area of infiltration seen in the previous film) a cavity of more than 3·1 centimetres (one and a quarter inches) in diameter, in which the lipiodol was scattered apparently mixed with retained secretion. On six occasions the sputum yielded no growth on attempted culture; it had since been injected into a guinea-pig. On no occasion was high temperature detected in the morning, and the pulse rate averaged 66. The evening temperature was, however, found to rise to about 37·8° C. (100° F.), the maximum being 38·9° C. (102° F.). The patient gained 3·15 kilograms (seven pounds) in weight in five weeks. The Wassermann test yielded no reaction. A twenty-four hours' specimen of urine contained no pus and no tubercle bacilli were found. The blood sedimentation test showed an abnormal but quiescent curve, the sedimentation index being 22 millimetres. Blood was sent for submission to the Vernes test, but the specimen proved unsuitable. The patient was referred to the Tuberculosis Bureau, where he was given an injection of 0·0005 cubic centimetre of albumose-free tuberculin, to which he gave a definite reaction. The discussion was concerned with the ætiology of the lesion, the consensus of opinion being against its tuberculous origin.

(To be continued.)

MEDICO-POLITICAL.

The Honorary Secretary of the Queensland Branch of the British Medical Association has forwarded the following statement for publication.

Proposed Representative Body.

The desirability of forming a Representative Body akin to the Representative Body of the Association in England has been under discussion by the Council of the Queensland Branch of the British Medical Association, and the Federal Committee is being asked to consider the matter at its next meeting. It is suggested that the proposed Representative Body should hold a business meeting to synchronize with sessions of medical congresses. Such a body would provide an opportunity for members throughout the Commonwealth to confer on general matters affecting the profession and on which it is of the utmost importance that a consolidated opinion should be formed.

NOMINATIONS AND ELECTIONS.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Akeroyd, John Finch, M.B., B.S., 1932 (Univ. Melbourne), Ballarat Base Hospital, Ballarat.

Bowen, William Fenton, M.B., B.S., 1932 (Univ. Melbourne), District Hospital, Warrnambool.

Morgan, Francis Patrick, M.B., B.S., 1929 (Univ. Melbourne), c.o. E. S. and A. Bank, 5, Gracechurch Street, London.

The Royal Australasian College of Surgeons.

ELECTION OF FELLOWS AND JUNIOR MEMBERS.

At a meeting of the Council of the Royal Australasian College of Surgeons, held in Sydney on September 3, 1932, the following candidates were elected as Fellows.

New South Wales: C. L. Chapman, H. M. de Burgh, J. G. W. Hill, K. L. H. Kirkland, S. F. Lynch, E. McA. Steel, J. Grenville Waine, C. E. Winston.

New Zealand: W. M. Brown, E. Ewart Brown, W. F. Buist, J. R. Connolly, H. K. Corkill, E. H. M. Luke, W. J. Macdonald, R. B. Martin, E. R. Reay, W. S. Robertson.

Queensland: Harold Crawford, G. F. Meade, H. W. Savige.

South Australia: Brian F. Moore.

Victoria: J. G. Winter Ashton, E. G. Dermer, A. M. Lazarus.

The following candidates for junior membership were elected:

New South Wales: E. J. Egan.

South Australia: Ian A. Hamilton.

Post-Graduate Work.

POST-GRADUATE COURSE IN MELBOURNE.

The Melbourne Permanent Post-Graduate Committee announces that the annual refresher course will be held from November 7 to November 18, 1932. The following syllabus has been issued:

Monday, November 7, 1932.

9.30 a.m. to 11 a.m.—Dr. W. G. D. Upjohn: "Empyema and Pulmonary Sepsis", Melbourne Hospital.

11.15 a.m. to 12.45 p.m.—Dr. S. O. Cowen: "Anæmia in Women of Middle Age", Melbourne Hospital.

2.15 p.m. to 3.30 p.m.—Dr. J. R. Williams: "The Treatment of Diabetes from a New Aspect", Melbourne Hospital.

3.30 p.m. to 5 p.m.—Dr. Wright Smith: "The Performance of an Autopsy Examination", Melbourne Hospital.

8.30 p.m.—Dr. C. H. Kellaway: Lecture I, "Acidæmia and Alkalæmia".

Tuesday, November 8, 1932.

9.30 a.m. to 11 a.m.—Dr. H. B. Devine: "Appendicitis: Its Diagnosis and Management. How to Improve Our Results" (lantern lecture), Saint Vincent's Hospital.

11.15 a.m. to 12.45 p.m.—Dr. Frank Niall: "Diagnostic Difficulties in Diseases of the Chest", Saint Vincent's Hospital.

2.15 p.m. to 3.30 p.m.—Dr. R. L. Forsyth: "Pneumonia in Infants and Children", Children's Hospital.

3.30 p.m. to 5 p.m.—Dr. Charles Osborn: "Common Deformities in Childhood", Children's Hospital.

8.30 p.m.—Dr. C. H. Kellaway: Lecture II, "Shock".

Wednesday, November 9, 1932.

9.30 a.m. to 11 a.m.—Professor Marshall Allan: "The Influence of Age on Parturition", Women's Hospital.

11.15 a.m. to 12.45 p.m.—Dr. Arthur Sherwin: "Pelvic Infections Following Parturition", Women's Hospital.

2.15 p.m. to 3.30 p.m.—Dr. John Tait: "Carcinoma of the Prostate", Melbourne Hospital.

3.30 p.m. to 5 p.m.—Dr. L. J. Clendinnen: "Results of Radium Therapy", Melbourne Hospital.

8.15 p.m.—B.M.A. clinical meeting, Children's Hospital.

Thursday, November 10, 1932.

9.30 a.m. to 11 a.m.—Dr. Fay Maclure: "Hints in Minor Surgical Technique", Alfred Hospital.

11.15 a.m. to 12.45 p.m.—Dr. J. F. Mackeddie: "Pains in the Legs", Alfred Hospital.

2.30 p.m. to 5 p.m.—Demonstration of infectious diseases patients by Dr. F. V. Scholes and staff, Infectious Diseases Hospital, Fairfield.

Friday, November 11, 1932.

9.30 a.m. to 11 a.m.—Dr. Julian Smith, Junior: "Colles's Fracture", Melbourne Hospital.
11.15 a.m. to 12.45 p.m.—Dr. Keith Fairley: "Medical Aspects of Goitre", Melbourne Hospital.
2.15 p.m. to 3.30 p.m.—Dr. Keith Colquhoun: "Skin Diseases Commonly Met With in General Practice", Saint Vincent's Hospital.
3.30 p.m. to 5 p.m.—Dr. John O'Sullivan: "Some Aspects of Clinical Radiology", Saint Vincent's Hospital.
8.30 p.m.—Dr. C. H. Kellaway: Lecture III, "Anoxæmia and Cyanosis".

Saturday, November 12, 1932.

9.30 a.m. to 11 a.m.—Dr. R. P. McMeekin: "Diagnostic Aspects and Treatment of Head Injuries", Melbourne Hospital.

Monday, November 14, 1932.

9.30 a.m. to 11 a.m.—Dr. Leslie Hurley: "Some Points in Diagnosis and Treatment: Advanced Cases of Diabetic Coma, Quinidine in Auricular Fibrillation, Idiopathic Secondary Anæmia, Salyrgan, Agranulocytosis", Melbourne Hospital.
11.15 a.m. to 12.45 p.m.—Dr. Edgar King: "The Diagnosis, Prognosis and Treatment of Ovarian Cysts", Melbourne Hospital.
2.30 p.m. to 5 p.m.—Demonstration at the Baker Institute, Alfred Hospital.
8.30 p.m.—Dr. C. H. Kellaway: Lecture IV, "Cardiac Failure".

Tuesday, November 15, 1932.

9.30 a.m. to 11 a.m.—Dr. W. S. Newton: "Bronchiectasis", Alfred Hospital.
11.15 a.m. to 12.45 p.m.—Dr. Balcombe Quick: "Acute Upper Abdominal Pain", Alfred Hospital.
2.30 p.m. to 5 p.m.—Demonstration at the Eye and Ear Hospital.

Wednesday, November 16, 1932.

9.30 a.m. to 11 a.m.—Dr. John S. Green: "The Toxæmias of Pregnancy", Women's Hospital.
11.15 a.m. to 12.45 p.m.—Dr. W. G. Cuscaden: "The Use of Radium in Gynæcology", Women's Hospital.
2.30 p.m. to 4 p.m.—Dr. J. Bell Ferguson: "Tuberculin in Diagnosis and Treatment", Central Tuberculosis Bureau.
8.30 p.m.—Dr. C. H. Kellaway: Lecture V, "Intestinal Obstruction".

Thursday, November 17, 1932.

9.30 a.m. to 11 a.m.—Dr. J. W. Grieve: "Rheumatic Carditis in Children and Adults", Saint Vincent's Hospital.
11.15 a.m. to 12.45 p.m.—Dr. Gordon Shaw: "Diseases of Joints", Saint Vincent's Hospital.
2.15 p.m. to 3.30 p.m.—Dr. J. G. Whitaker: "Bone Diseases in Children", Children's Hospital.
3.30 p.m. to 5 p.m.—Dr. W. W. McLaren: "Digestive Disturbances in Babies", Children's Hospital.

Friday, November 18, 1932.

9.30 a.m. to 11 a.m.—Dr. Henry Searby: "Tumours in the Neck", Melbourne Hospital.
11.15 a.m. to 12.45 p.m.—Dr. Ivan Maxwell: "Disturbances of Gastric Secretion", Melbourne Hospital.
8.30 p.m.—Dr. C. H. Kellaway: Lecture VI, "Some Disorders of Calcium Metabolism".

The fee for the course will be three guineas. Accommodation will be available for a limited number of graduates at the Melbourne and Alfred Hospitals at an additional fee of three guineas per week. Those wishing to take advantage of this provision are asked to notify the Honorary Secretary of the Committee, 61, Collins Street, Melbourne, as soon as possible.

It should be noted that the series of lectures by Dr. C. H. Kellaway constitutes a separate course.

University Intelligence.

THE UNIVERSITY OF MELBOURNE.

Medical Research Funds Committee.

THE University of Melbourne has established a "Medical Research Funds Committee" for the purpose of advising the Council in the administering of all funds available for medical research. The Committee desires to encourage gifts for research without restrictions as to their application to any particular line of research; and, where a donor takes special interest in any particular branch of medical science, to offer advice regarding the best way of defining any special restriction which is desired. In either case the University undertakes at all times to endeavour to attach the donor's or testator's name to any scholarship awarded out of such funds. It is felt that in the past there has been a lack of real value in some bequests for medical research by reason of the fact that the donors have acted with insufficient information. Bequests may thus be robbed of much of their usefulness.

It is hoped that bequests and donations may be attracted by the new committee, which is representative of all branches of medical science, so that it may have funds available to assist any young graduate who may show capacity and desire to undertake an approved piece of research work.

It is felt that it is in this way that research funds can be used to the greatest advantage, although, as before stated, if donors specially desire that their trust money should be spent in any specified way, such wishes will always be carefully carried out.

It is therefore hoped that friends of the University who desire to further the cause of medical research will make their bequests available for medical research in general, free of all limiting conditions, with, if desired, the attachment of the donor's name to any piece of research carried out with such funds.

The Committee has at present the administration of the Stewart bequest, the Alwyn Stewart and Alcock bequests and the clinical research fund, and will claim an interest in the general research funds of the University.

The Committee consists of the Chancellor, Vice-Chancellor, and Chairman of the Finance Committee of the University, the Professors of Anatomy, Physiology, Pathology and Obstetrics, the Director of the Bacteriology Department, the Lecturers in Medicine and Surgery, the medical members of the University Council, and two clinical teachers, and it is hoped that a committee so constituted will commend itself to those who desire to aid medical research, as one that can be relied on to insure that gifts will be used to the best advantage.

The Committee suggests to medical men to whose notice this statement comes, that they should take opportunities of bringing the matter to the attention of trustee companies, solicitors and others likely to be interested in gifts to the University for medical research.

Correspondence.

THE PSYCHOPATHOLOGY OF CHILDHOOD.

SIR: Your issue of September 3 gives a report, evidently a condensed report, of Professor Dawson's paper, "The Psychopathology of Childhood".

In your report the following sentence occurs: "Closely allied to the treatment of bad habits . . ."

Whilst finding Professor Dawson's paper of great interest, I think that the use of the word "bad" is likely to lead to confusion and further faulty thinking. Those who make a study of medical psychology will no doubt not be led into error by this word, knowing that it is used to mean faulty; but to most people the word bad conveys an ethical or moral meaning.

In so far as the medical man uses words with a moral implication, he becomes something else than a medical

man and therefore loses his position as a scientific interpreter of mental phenomenon.

After all, many mental disorders originate broadly in a conflict between the demands of morality and the instinctual demands of the body. I take it that it is the medical man's problem to resolve these conflicts, as far as he is able, by setting free the demands into channels appropriate to the individual and his environment.

It is not his province to increase the force of repression, which is already being done by each one's own super *ego* and which the use of such words as bad will only modify in a harmful direction.

It may seem a small thing to cavil at, but there is a growing tendency amongst certain cults of psychology to advocate moralizing to the suffering patient, who in the majority of cases of mental disorder is already suffering from an excess of "guilt".

Yours, etc.,

PAUL G. DANE.

86, Collins Street,
Melbourne,
September 8, 1932.

SQUINT AND THE ORTHOPTIC CLINIC.

SIR: In your timely article on the above subject you express the hope that an orthoptic clinic will be established in Sydney. I write to inform you that such a clinic is in process of formation at the Royal Alexandra Hospital for Children, at my suggestion. We are only awaiting the arrival of the most modern instrument, the synoptoscope. We expect the clinic to be in working order before the end of the year. I willingly acknowledge indebtedness to Dr. Ringland Anderson, of Melbourne, for his introduction to, and advocacy of, this new instrument for fusion training.

Yours, etc.,

E. TEMPLE SMITH.

141, Macquarie Street,
Sydney,
September 23, 1932.

SQUINT AND SIGHT SAVING.

SIR: Those interested in the question of squint and its treatment would welcome the editorial which appeared in the issue of this journal dated September 24. So much development has taken place in the non-operative treatment of this disorder in the last three years, that it is most suitable that such a clear statement should appear. If parents are told by medical men that their squinting children can receive no treatment till they are seven, it is surely the fault of oculists. It suggests that very inadequate training of students and that a tendency amongst oculists to be satisfied with operative correction are causes of such a belief.

Much can be learned of any poorly understood disorder if attention is given to it in a special clinic. The formation of a sight saving class in any hospital will soon prove to its promoters how little is really understood concerning squint. The first of such classes in England to be formed was that at the Westminster Hospital; Moorfields followed soon afterwards. The work of Maddox, of Bournemouth, was probably the greatest factor in initiating this movement, and the work is now under the able leadership of Miss Maddox. Such work must be regarded as a form of research, for new problems are constantly arising. Such a class constitutes a very useful follow-up scheme, so that remote effects of treatment can be studied. When specially gifted lay assistants have been trained in such a class, they can become of great assistance to oculists by commencing in private practice. Three such workers have been in Collins Street for over a year.

It is wise when starting such a class to limit the patients to those who have at least $^6/_{12}$ in the squinting eye and, in proving this, the practitioner must make sure that the visual test is not simply a demonstration of a child's good memory. Fusion training has been started in several cases and has been futile, because amblyopia has been masked by the child's memory of the letters of the test type. It is wise also to exclude at first children who were born with one eye turned and those who have a true alternating squint. In these progress is very slow, for the establishment of an abnormal relationship between the two retinæ is a serious obstacle. It is economical to operate on those who cannot attend twice weekly or those with an exceptionally large squint, and then insure the development of stereoscopic vision by training.

The prevention and the treatment of amblyopia and of an abnormal retinal correspondence demand the earliest possible attention. This will only be possible when practitioners and parents realize that the oculist should see the child as soon as the squint develops.

Yours, etc.,

J. RINGLAND ANDERSON.

108, Collins Street,
Melbourne,
October 5, 1932.

POLIOMYELITIS.

SIR: In your issue of September 17, Dr. Karen Helms states that a *post mortem* examination of certain cases reported by Dr. Steigrad discloses the following fact. Those instances in which a diagnosis of poliomyelitis was made and the cerebro-spinal fluid cell count was not raised, are proved to have been mistaken diagnoses. If this is so, then, of course, one of the premises in the argument contained in my report to your journal is false, and I withdraw my conclusion. I would, however, venture to suggest that if Dr. Steigrad would now publish further details of his cases, he would be rendering a very valuable service to all interested in infantile paralysis.

In conclusion, I wish to deny Dr. Helms's insinuation that my original article was a "claim of cure in a doubtful case". It was simply reported because I thought it belonged to a series of unusual cases of poliomyelitis (such as Dr. Steigrad reported) in which the cell count was normal. Far from the result being to "promulgate inaccurate ideas", the correspondence has corrected a misconception on my part and has, I trust, proved of interest to other readers.

Yours, etc.,

S. GOLDBERG.

Griffith,
New South Wales,
September 28, 1932.

Obituary.

AUGUSTUS JOSEPH WALFORD PETTIGREW.

WE regret to announce the death of Dr. Augustus Joseph Walford Pettigrew, which occurred on September 30, 1932, at Balwyn, Victoria.

SYBIL JEAN HAWKINS.

WE regret to announce the death of Dr. Sybil Jean Hawkins, which occurred on September 30, 1932, at Bundoora, Victoria.

HAMPDEN CARR.

WE regret to announce the death of Dr. Hampden Carr, which occurred on September 30, 1932, at Kangaroo Island, South Australia.

PATRICK SHAW.

WE regret to announce the death of Dr. Patrick Shaw, which occurred on October 4, 1932, at Ballarat, Victoria.

MARGARET McLORINAN.

WE regret to announce the death of Dr. Margaret McLorinan, which occurred on October 9, 1932, at Melbourne, Victoria.

THE ROBERT H. TODD PRIZE IN MEDICAL JURISPRUDENCE.

ADDITIONAL subscriptions towards the Robert H. Todd Prize in Medical Jurisprudence have been received as follows :

£2 2s.: Dr. M. Archdall.

£1 1s.: Dr. B. P. Anderson-Stuart, Dr. A. L. Dawson, Dr. Muriel McIlrath, Dr. A. M. Purves.

The total amount received to date is £82 14s.

Books Received.

A SHORTER SURGERY: A PRACTICAL MANUAL FOR SENIOR STUDENTS, by R. J. McNeill Love, M.B., M.S., F.R.C.S.; Third Edition; 1932. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 422, with 96 illustrations. Price: 16s. net.

PHYSIOLOGY OF THE CENTRAL NERVOUS SYSTEM AND SPECIAL SENSES (FOR THE USE OF STUDENTS), by N. J. Vazifdar, L.M. and S., F.C.S., F.C.P.S.; Fifth Edition; 1929. Calcutta: Thacker, Spink and Company. Demy 8vo., pp. 301, with 30 illustrations. Price: 7s. net.

HEART DISEASE: THE PRINCIPLES OF DIAGNOSIS AND TREATMENT, by C. Bramwell, M.A., M.D., F.R.C.P.; 1932. London: Edward Arnold and Company. Demy 8vo., pp. 251, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

OCT. 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 19.—Western Australian Branch, B.M.A.: Branch.
OCT. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 26.—Victorian Branch, B.M.A.: Council.
OCT. 27.—South Australian Branch, B.M.A.: Branch.
OCT. 27.—New South Wales Branch, B.M.A.: Branch.
OCT. 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. H. McLaren has been appointed Government Medical Officer at Cowra, New South Wales.

* * *

Dr. H. R. Hill (B.M.A.) has been appointed Government Medical Officer at Culcairn, New South Wales.

* * *

Dr. E. V. Keogh has been appointed Government Medical Officer under the provisions of Section 3 of *The Miners' Phthisis Act*, 1922, Western Australia.

* * *

Dr. J. G. Drew (B.M.A.) has been appointed Deputy Commissioner of Public Health, Queensland, pursuant to the provisions of *The Health Acts*, 1900 *to* 1931.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officer.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.

VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.